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AMERICAN JOURNAL OF OPHTHALMOLOGY

VOLUME 23 APRIL, 1940 NUMBER 4

CORNEAL TRANSPLANTATION*

A CRITICAL REVIEW OF SIXTEEN CASES

J. WESLEY MCKINNEY, M.D.
Memphis, Tennessee

The substitution of transparent tissue for the opacified cornea has been attempted for more than a century, yet not until recent years have these efforts been rewarded with consistent success. The early attempts to substitute glass for the opaque cornea, though interesting, were fatal for the eye. Total keratoplasties, in which the entire cornea was transplanted, led to a similar outcome: the transplants became opaque, the eyes developed glaucoma, and, in the majority, phthisis bulbi ensued. Lamellar keratoplasties, wherein only a part of the thickness of the cornea was transplanted, were then undertaken. These were considered safer than the penetrating type in that the eye was not opened. A scar always formed, however, between the graft and the underlying cornea.

Partial penetrating keratoplasty, or removal of a portion of the full thickness of the cornea and its substitution by a similar portion of clear cornea, is now accepted as the procedure of choice. The technique of this operation has been developed from animal experiments and from observations on the human subject.

In this paper it is the author's purpose merely to present his first 16 cases in which keratoplasty was performed and to record certain observations therefrom. The patients have been followed for periods varying from six months to two-and-one-half years after operation. In order to determine the suitability of leucomatous eyes for transplantation, many were operated upon which seemed from previously recorded observations to offer poor prognoses.

CASE REPORTS

Case 1. M. W., a female, aged 35 years, was first seen on January 4, 1937. According to her history, she had had sore eyes as a child; she had since been practically blind in the right eye and vision in the left had been greatly impaired.

On examination of the right eye, vision sufficed to count fingers at three feet. The cornea was slightly ectatic and involved in the leucoma throughout; the scarring was only moderately dense, however, and it was believed that probably some corneal elements were present over its entire surface. Above was an area of relatively clear cornea, through which the iris could be seen. Beneath this area the iris was free, but was elsewhere adherent to the cornea. The tension was normal and light projection good.

In the left eye, vision was 6/60. There was dense scarring about the periphery of the cornea, and its lower one third was leucomatous. The pupil was almost totally occluded; only a small aperture remained, through which the fundus reflex could be seen. The tension was normal.

*From the University of Tennessee College of Medicine, Department of Ophthalmology, and the Memphis Eye, Ear, Nose, and Throat Hospital.

A corneal transplantation was performed on the right eye on January 12, 1937. The square segment was so excised as to permit one side of the graft to be contiguous to the relatively clear cornea. A large portion of the adherent area of the iris was resected with the segment; the remaining adhesions were freed with the spatula. Below, the cornea was twice the normal thickness. On removal of the square, an anterior polar cataract was discovered, and, in addition, the cortex was found to be partially cataractous, allowing only a faint red reflex.

The postoperative course was uneventful. The graft remained clear; only a slight edema developed. The anterior polar cataract could be seen at all times. During the first month, interstitial infiltration with accompanying fine, deep blood vessels appeared around the edges of the graft; this process, however, did not reach the center. A fine epithelial vesiculation extending over the cornea for a distance of 1 or 2 mm. beyond the graft was also observed. During the second month, the interstitial infiltration and vesiculation cleared, leaving the periphery of the graft hazy, while the center remained clear.

At the end of two months the patient's vision was 4/200. She was advised to have the lens removed from the right eye, but in view of the fact that vision in the left eye had been improved to 6/15 and Jaeger 4 by optical iridectomy, further surgery was refused. In January, 1939, the left eye was lost from gonorrheal ophthalmia. On May 14, 1939, an intracapsular cataract extraction was performed on the right eye. The resulting vision equaled 6/60 and Jaeger 1 held close to the eye (fig. 1).

The donor eye was removed on the day of the transplantation. The cornea was normal except for a small epithelioma at the limbus. Two grafts were taken, one to be used in case 2.

Case 2. O. W., a female, aged 25 years, was first seen on December 27, 1936. She had been blind in the left eye for five years, following injury from a firecracker explosion.

Examination of the left eye disclosed a horizontally oval dense scar in the center of the cornea, to which the iris was everywhere adherent except over a small area below. The periphery of the cornea was clear. There was no anterior chamber. The tension was normal, and light projection was good. Vision was reduced to the perception of moving objects.

The right eye was normal both internally and externally. Vision was 6/5.

Corneal transplantation was performed on the left eye on January 12, 1937. The square was so excised as to permit one side of the graft to be contiguous below to relatively normal cornea. The cornea was at least three times the normal thickness at the scar. A portion of the iris was elevated with the corneal segment, and the remaining adherent iris was freed with the spatula. On removal of the square, the fundus reflex was found to be rather dim, as a result of an anterior capsular and posterior cortical opacity.

The postoperative course was uneventful. The graft remained clear at all times; there was little edema (fig. 2). A fine epithelial vesiculation appeared on and immediately surrounding the graft and persisted for five months. Only a slight amount of interstitial infiltration and almost no deep vascularization could be detected. Vision was improved to only 3/60. Removal of the lens at a later date was advised.

The patient was not seen again until November 1, 1937. She stated that the eye had been red and painful for 10 days. The graft was slightly hazy from edema, and the lens was totally opaque. The tension was 2 plus, and failed to respond to miotics after 16 days. The lens was then removed by combined extracapsular ex-

traction, through a keratome incision. The graft rapidly cleared, becoming almost as clear as a normal cornea by the eleventh postoperative day. A small portion of remaining cortex was soon absorbed. The fundus was plainly visible and appeared normal. The best corrected vision, however, was 4/60.

Case 3. E. G. H., a male, aged 49 years, was first seen on June 18, 1937. He gave a history of having been shot in the right eye with a gun in November, 1934. Vision had been immediately lost, but had improved to some extent after a few months. A severe ulcer had developed in the cornea of the left eye in 1936, necessitating hospital treatment for three months.

The cornea of the left eye was involved in a dense leucoma which contained several large blood vessels. Only a small area of the cornea above and nasally remained relatively clear, and this was twice the normal thickness. The iris was seen through the clear area, but could not be dilated; on transillumination, it was found adherent throughout to the cornea. The tension was normal, but light projection was impaired. Vision equaled light perception.

On examination of the right eye, the anterior chamber was found to be normal; the retina, however, was completely detached and atrophic.

The superficial blood vessels in the leucoma were obliterated by diathermy on September 24, 1937, and on October 10, 1937, a corneal transplantation was performed. The cornea was at least three times the normal thickness, and the scarred iris was adherent throughout. The lens, which was densely cataractous, was removed through the corneal opening. The posterior capsule was left intact.

There was little postoperative reaction. On the third day the sutures became detached at intervals, and on the following day a slight gap, with some bleeding, was observed on two sides of the graft. Seidel's sign was negative. The graft remained only fairly clear and rather edematous during the postoperative period. After one month, the vision was 2/200 with a plus 11.00 sphere (fig. 3). On February 23, 1938, the patient stated that he could not see so well as formerly. The graft was edematous, tension was 52 mm. Hg, and vision with a plus 11.00 sphere was only 1/200. Since myotics failed to control the tension, a cyclodialysis was performed on March 15, 1938. The graft, however, continued to opacify and, when the patient was last seen, on September 27, 1938, was only slightly less opaque than the surrounding cornea.

The donor eye, which contained a sarcoma of the choroid, was enucleated immediately prior to transplantation. Another graft from this cornea was used in case 4.

Case 4. J. S., a male, aged 58 years, was first seen on July 21, 1937. Vision had been failing in both eyes for 10 or 12 years, from an indefinite cause. A combined extraction in the capsule had been performed several months earlier.

The cornea of the left eye was involved in a moderately dense leucoma throughout. The iris could be seen with operative coloboma above; there was a faint fundus reflex. The anterior chamber was normal. Tension was normal, and light projection was good. Vision was best with a plus 10 sphere, equaling counting fingers at two feet.

Examination of the right eye revealed a total leucoma, a shrunken globe, and only faint light perception.

Corneal transplantation was performed on October 20, 1937. Immediately upon opening the cornea, a large amount of vitreous escaped. The graft was put in place with extreme difficulty, and the approximation of the wound edge was grossly inaccurate.

The postoperative course was marked

by progressive scarring of the graft. The resulting vision was the ability to discern moving objects.

The donor eye had been removed six hours previously, for sarcoma of the choroid.

Case 5. R. E. L. J., a male aged 75 years, was first seen on May 21, 1937. He gave a history of having had trachoma since 1887, and of having been unable to read for many years. He was a diabetic, though the disease was under control.

On examination of the right eye, many turned-in lashes were observed on the upper and lower lids. The conjunctiva was scarred throughout, and the fornices were practically obliterated. The entire conjunctiva and corneal epithelium had a dry appearance; tests with litmus paper showed no tears. The corneal leucoma contained a number of large superficial blood vessels. The iris was faintly visible; the pupil was centrally situated. The anterior chamber was of normal depth. Tension was normal, and light projection good. Vision was the ability to discern moving objects.

Examination of the left eye also revealed many turned-in lashes. The conjunctiva, however, was scarred to a less degree than that of the right eye. The cornea was superficially nebulous and contained fine superficial vessels. The pupil was round and active. The fundus could be faintly seen and appeared normal. Vision was 3/60, and the tension, normal.

The inturned lashes on the right eyelid were removed, the superficial vessels on the cornea were obliterated, both by diathermy, and a basal iridectomy was carried out preliminary to keratoplasty.

Corneal transplanation was performed on the right eye on November 19, 1937. On the second day the graft became infiltrated, and an abundant purulent conjunctival discharge appeared. On the third day, a dense yellow infiltration spread over the entire graft. Ultimately, the graft was lost and the lens extruded; thereafter, the corneal opening gradually closed and the globe became shrunken.

The donor eye was enucleated six hours previously, for sarcoma of the choroid. This donor was also used for case 6.

Case 6. S. W., a female, aged 66 years, was first seen on October 11, 1937. The left eye had been blind since childhood, as a result of a blow from a broom. Vision in the right eye had been limited to light perception for at least 25 years, from an unknown cause.

On examination of the right eye, the cornea was found to be densely leucomatous throughout, and slightly ectatic; some calcification was present below. The iris was not visible through the cornea, but on transillumination the pupil proved to be round and centrally situated, and to react readily. The anterior chamber was of normal depth. The tension was normal and light projection good. Vision was light perception only.

The appearance of the left eye was similar to that of the right. There was no anterior chamber, however, and no light perception.

Corneal transplantation was performed on the right eye on November 18, 1937. The pupil was constricted with eserine preoperatively; on removal of the corneal segment, it was found to be small and centrally located. The cornea was twice the normal thickness. At the close of the operation, atropine and suprarenin ointments were instilled to dilate the pupil.

During the early postoperative period the edema was of only slight degree and the graft remained clear; the iris was at all times plainly visible. During the second and third weeks, infiltration and deep vascularization slowly progressed, though not involving the center of the graft. Roentgen-ray therapy was begun at this

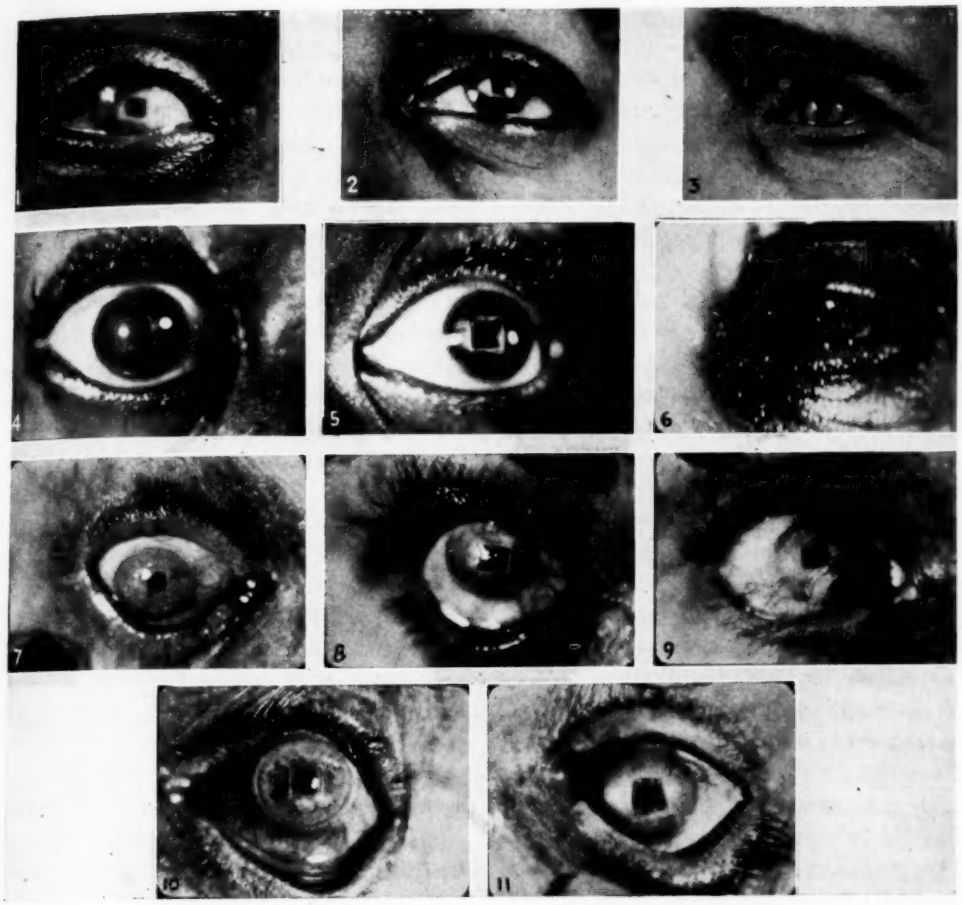

Fig. 1 (McKinney). Case 1. Photograph taken two months after keratoplasty.
Fig. 2 (McKinney). Case 2. Photograph taken two months after keratoplasty.
Fig. 3 (McKinney). Case 3. Photograph taken one month after keratoplasty. This graft subsequently became opaque as a result of glaucoma.
Fig. 4 (McKinney). Case 8. Photograph taken 18 months after keratoplasty.
Fig. 5 (McKinney). Case 9. Photograph taken 10 months after keratoplasty.
Fig. 6 (McKinney). Case 10. Photograph taken one month after keratoplasty. The graft subsequently became entirely opaque.
Fig. 7 (McKinney). Case 11. Photograph taken four months after keratoplasty.
Fig. 8 (McKinney). Case 13. Photograph taken four months after keratoplasty.
Fig. 9 (McKinney). Case 14. Photograph taken six months after keratoplasty.
Fig. 10 (McKinney). Case 15. Photograph taken three months after keratoplasty.
Fig. 11 (McKinney). Case 16. Photograph taken four months after keratoplasty.

time and carried out in weekly treatments of approximately 120 roentgens each. On February 10, 1938, the graft was much clearer and the iris could again be seen; vision, however, had diminished to faint light perception. Three weeks later the graft was only slightly opaque and contained a few fine, deep vessels. The lens was diffusely opaque, and light perception had been lost. The patient died of cerebral hemorrhage before her next scheduled visit.

The donor eye was enucleated, immediately prior to transplantation, for sarcoma

of the choroid. The same eye was used for case 5.

Case 7. C. S., a male, aged 19 years, was first examined on December 14, 1937. According to his history, a firecracker had exploded in his left eye five years earlier. The injury had been followed by a painful ulcer which persisted for two months.

The left eye contained a dense central leucoma involving one third of the cornea above. The cornea surrounding the leucoma was relatively clear. The leucoma bulged moderately and the iris was everywhere adherent. The tension was elevated to fingers, and light projection was good. Vision equaled light perception.

The right eye was normal both internally and externally. Vision was 6/60 and Jaeger 1.

Corneal transplantation was performed on January 10, 1938. The graft was placed eccentrically, so as to be continguous to the relatively normal cornea. A portion of the adherent iris was removed with the square segment, and the remainder was freed from the cornea with the spatula. The cornea was three times the normal thickness on three sides. The anterior capsule of the totally opaque lens was opened with the keratome and the lens material was expressed through the corneal defect.

The graft was extremely edematous at the first dressing. A white membrane soon formed on its posterior surface, the graft became vascularized and partially opaque, tension was elevated, and large bullae appeared in the epithelium. Five months after operation the graft was moderately opaque throughout and slightly bulging, and a dense membrane was present posteriorly. The tension was 3 plus, and vision equaled light perception.

The donor eye had been enucleated immediately before transplantation, for sarcoma of the choroid.

Cases 8 and 9. W. C., a male, aged six years, was first seen on January 6, 1937. He had had poor vision in both eyes following an inflammatory condition, presumably interstitial keratitis, two years previously.

Examination of the right eye disclosed a fine, deep vascularization of the cornea and diffuse interstitial opacities, the latter being most pronounced in the center. The pupil was active and centrally located. After dilatation of the pupil, the fundus was visible, although no details could be defined. Tension was normal. Vision sufficed only for counting fingers at two meters.

The cornea of the left eye was similar to that of the right; the opacity, however, was less dense. The pupil was active and centrally located. After dilatation of the pupil, the details of the fundus could be seen indistinctly and appeared to be normal. Tension was normal, and vision equaled 6/60.

Following antisyphilitic treatment over a period of four months, corneal transplantation was performed on the right eye on May 17, 1937. The pupil was constricted preoperatively with eserine, and at the termination of the operation atropine and suprarenin ointments were instilled. The cornea was of normal thickness.

At the first dressing the pupil was widely dilated and the anterior chamber had re-formed. The graft was edematous and deeply vascularized. This condition cleared slowly during the next several months. On October 4, 1937, diffuse nebulae were observed in the stroma, particularly just in front of the endothelium, and the deep blood vessels were found to have shrunken to fine threads. The fundus details could be hazily distinguished. Vision equaled 6/60 and Jaeger 1 with difficulty four inches from the face.

Corneal transplantation was performed

on the left eye on March 23, 1938. The pupil was constricted with eserine preoperatively, and postoperatively atropine and suprarenin ointments were instilled.

At the first dressing, on the following day, the pupil was widely dilated and the anterior chamber had re-formed. The graft remained clear; there was little edema. On the fifteenth postoperative day the fundus was visible through the graft. Ten days later the graft had become slightly opaque throughout and of twice the normal thickness. Two weeks later a narrow zone of stromal infiltration and vascularization could be seen around the periphery; otherwise, the graft had cleared, and again the fundus was faintly visible. Thereafter, the graft continued to clear, and on June 10, 1938, contained only a few indistinct nebulae. Vision was 6/15 and Jaeger 1, and has remained so to the present time (figs. 4 and 5).

The donor eye for the first transplant was enucleated for glioma of the retina, immediately prior to operation. For the second transplant, the donor eye was enucleated for sarcoma of the ciliary body.

Case 10. R. L. W., a male, aged 52 years, was first seen on March 28, 1938. He gave a history of having had sore eyes as a child. An optical iridectomy had been carried out on the left eye in 1900, and the right eye had been removed in 1912. In 1934, a cataract was extracted from the left eye, and this was followed by a needling.

On examination of the left eye, a nystagmus of rather wide excursion was discovered. The cornea was densely leucomatous, only a small area above being relatively clear. Through this area the iris was seen dimly and was everywhere adherent except at the site of the iridectomy. A faint red reflex could be detected above. Tension was normal and light projection fair. Vision sufficed only to discern moving objects.

Corneal transplantation was performed on May 4, 1938. The fibrotic iris and posterior capsule of the lens were adherent to the cornea; the portion attached to the corneal square was excised. A small amount of vitreous was lost.

The postoperative reaction was of moderate degree. The sutures were removed on the seventh day. On the following day a slight gaping appeared between the graft and cornea on the upper side. Seidel's sign was negative, however, and the graft remained fairly clear. On the 17th day, a membrane was observed behind the graft, and within this membrane was a small opening through which a faint red reflex could be seen (fig. 6). Two months after the operation the graft had become somewhat cloudy; the entire cornea was flattened, and the opening in the membrane behind the graft had closed. Four months postoperatively the graft, although again fairly clear, was slightly invaginated. The membrane behind the cornea was dense, and vision equaled light perception. The tension was low.

The donor eye was enucleated, immediately preceding the transplantation, for sarcoma of the choroid. Glaucoma had been present for three days prior to enucleation. A graft was also taken from this cornea for case 11.

Case 11. H. G., a female, aged 76 years, was first seen on December 12, 1936. She had had an uncomplicated intracapsular cataract extraction and peripheral iridotomy of the right eye six months previously. The corrected vision was 6/7.5 and Jaeger 1. Light perception in the left eye had been lost as a result of glaucoma. Vision began to fail on May 2, 1937.

On examination, all the layers of the cornea were opaque above, Descemet's membrane was wrinkled, and minute vesicles were observed in the epithelium. Vitreous filled the anterior chamber and

came in contact with the cornea, especially above. The margins of the iris were adherent to the vitreous. The pupil was small and round. Vision was 6/24 and tension 17.

Despite all treatment, the opacity progressed slowly in all layers, ultimately involving the entire cornea. On April 5, 1938, large epithelial bullae were present; the fundus could not be seen. Vision equaled the perception of moving objects.

Corneal transplantation was performed on May 4, 1938. Eserine was instilled prior to operation, and atropine and suprarenin ointments postoperatively. No vitreous was lost.

There was only a slight postoperative reaction. The sutures were removed on the sixth day. On the following day a small gap appeared at the temporal and lower sides of the wound; Seidel's sign, however, was negative. The graft was clear and there was a bright fundus reflex. One month after operation the graft was still clear and vision was 6/60 and Jaeger 14. One week later there was some interstitial opacity of the graft, though no vascularization. Fine epithelial vesicles were also apparent. After two additional weeks the interstitial opacity had cleared almost completely, and the corrected vision was then 6/60 and Jaeger 9; this status remained unchanged for several months (fig. 7). At the present time there are two blebs on the graft, and corrected vision is 6/120 and Jaeger 16.

The donor eye was enucleated, immediately prior to transplantation, for sarcoma of the choroid. Acute glaucoma had been present for three days. A graft was also taken from this cornea for case 10.

Cases 12 and 13. L. B. M., a female, aged 28 years, was first seen on May 19, 1937. Interstitial keratitis had developed in both eyes six years previously, and she had since been practically blind. Adequate antisyphilitic treatment had been administered.

Examination of the right eye revealed an opacity of the entire cornea, with deep and superficial vascularization. The iris was only faintly discernible; the pupil was active. No fundus reflex could be seen. The anterior chamber was of normal depth. The tension was normal and light projection was good. Vision was the ability to distinguish moving objects.

In the left eye there was a dense, bulging leucoma to which the iris was adherent throughout. The tension was 2 plus, and there was no light perception.

Corneal transplantation was performed on the right eye on October 6, 1937. Eserine was instilled preoperatively, and atropine and suprarenin ointment were instilled following operation. The cornea was twice the normal thickness above. The margins of the wound bled freely.

The postoperative reaction was rather severe; the corneal blood vessels became intensely injected, and an interstitial infiltration of the graft soon appeared. After one month the graft had cleared to some extent and vision equaled 1/200. Two weeks later, glaucoma developed and could not be corrected by medical treatment. An iridencleisis was performed on December 17, 1938. The tension was controlled for three months; thereafter, it again increased and could not be reduced medically. On March 8, 1938, a Lagrange sclerectomy was performed; this controlled the tension, but the graft was cloudy and vision declined to its former state, sufficient only to discern moving objects.

Another transplantation was carried out on August 30, 1938. The graft was so placed that two sides were within the previous graft. On removing the corneal square, the fundus, which was plainly visible through the opening, appeared normal. The reaction to this graft was less than that following the first, and subsided rapidly; the graft remained clear and there was little edema. Twenty days after

operation vision was 6/120. At that time, a marginal infiltration and vascularization of the graft were seen adjacent to the patient's cornea, though there was no infiltration from the two sides adjacent to the former graft. Two weeks later, vision was 6/30, and with pin hole 6/18—1 (fig. 8). During the following month the patient reported that vision became alternately better and worse. Tension was found to be 30 mm. Hg (Schiötz), but was readily reduced by massage. A thin interstitial haze, accompanied by fine vascularization, gradually spread through the graft from the sides abutting the cornea to the sides abutting the previous graft. Vision at this time is 6/30 and Jaeger 12.

For the first transplant, an eye enucleated for sarcoma of the choroid was used; for the second, the graft was taken from an eye, enucleated 30 hours previously, for glioma of the retina. The cornea of the latter was edematous as a result of a glaucoma of 10 days' duration. A second graft was taken from this cornea for transplantation in case 14.

Case 14. W. A. M., a male, aged 37 years, was first seen on May 27, 1938. He gave a history of having had sore eyes after birth and poor vision all his life. He had been educated at a school for the blind.

Examination of the right eye disclosed a diffuse, rather dense corneal opacity, with a superficially vascularized, dense, round scar occupying the lower half of the cornea. The iris was not visible, but on transillumination was found to be attached to the scar below, though free above, and to react slightly. The tension was normal and light projection good. Vision was the ability to discern moving objects. There was a nystagmus of wide excursion.

The cornea of the left eye was densely opaque throughout and appeared thinner than normal. There was no bulging. On transillumination, the pupil was vertically oval and apparently attached to the cornea below. Tension was unimpaired, and light projection good. Vision was reduced to discerning moving objects.

On June 1, 1938, the superficial blood vessels in the leucoma of the right cornea were obliterated by diathermy, and on August 30, 1938, a corneal transplantation was performed, eserine having been instilled previously. No iris was encountered in the wound, although it could be seen adherent to the cornea below. The lens was apparently normal. Atropine and supranenin were instilled postoperatively.

The reaction was of only moderate degree and the graft remained clear. After two weeks, a narrow tongue of deep infiltration was observed projecting from the nasal margin. During the fourth week the graft became mildly edematous; this, however, soon disappeared (fig. 9). At the present time a fine horizontal linear opacity in the deep stroma slightly clouds the fundus reflex, but the graft is otherwise clear. Vision is 2/200.

The donor eye was enucleated for glioma of the retina 30 hours previously. The cornea was edematous as a result of a glaucoma of 10 days' duration. A second graft from this cornea was used for case 13.

Case 15. G. A. K., a male, aged 69 years, was first seen on March 30, 1938. He stated that his vision had been failing in both eyes for 14 years. The condition had been diagnosed as corneal dystrophy. On several occasions the left eye had been inflamed because of mild central ulcers.

Examination of the left eye revealed an irregular central opacity in the superficial stroma of the cornea. In the center of this area were seen epithelial bullae, which diminished in number and size toward a clear periphery. The pupil was active and centrally situated. After wide dilatation of the pupil, the fundus was faintly visible and appeared normal. The tension was normal. Vision was 1/60

and Jaeger 19 held close to the eye.

The changes in the cornea of the right eye resembled those in the left, though less extensive. Vision was 1/60 and Jaeger 14 held close.

Corneal transplantation was performed on September 23, 1938. Eserine was instilled preoperatively, and atropine and suprarenin after operation.

The graft became so edematous as to seem to bulge forward. After 10 days, however, the edema subsided, leaving the graft no thicker than the surrounding cornea and only slightly cloudy. Three weeks after operation vision was 6/60. Although the graft remained normally clear, because of a central nuclear lens opacity vision was not improved beyond 6/60 and Jaeger 6 held close (fig. 10).

The donor eye had been enucleated, 24 hours previously, for an epithelioma at the limbus.

Case 16. J. T. K., a male, aged 75 years, was first seen on October 26, 1938. According to his history, he had had recurrent ulcers of both corneae for 15 years. Vision had become more impaired with each ulcer.

The cornea of the right eye was superficially opaque to a moderate degree throughout, though the opacity was most dense in the center. A fine superficial vascularization was apparent. The iris was faintly visible. The pupil was centrally located, but was small and reacted slowly. Vision was the ability to discern moving objects. Tension was normal, and light projection was good.

Examination of the left eye revealed a moderately dense superficial opacity in the center of the cornea; this faded at the periphery, leaving the cornea almost clear. The pupil was in the center, but was small and slow in reaction. Vision perceived only moving objects. Tension was normal, and light projection good.

A corneal transplantation was performed on December 14, 1938. The pupil was constricted with eserine preoperatively, and atropine and suprarenin were instilled postoperatively. While inserting the sutures, the needle perforated the cornea, the aqueous escaped, and the anterior chamber collapsed. It was therefore necessary to make the initial penetrating incision above with a small scalpel, rather than with the keratome. The cornea was only half the normal thickness.

The sutures were removed on the eighth postoperative day. The following day the graft was clear; there was little, if any, edema. The iris was plainly visible, and there was a bright fundus reflex. After a few days the fundus could be seen; it appeared normal. An incipient cortical cataract was detected. Vision improved rapidly, equaling 6/15 and Jaeger 3 with a +2.50 D.sph. four weeks following operation. The graft was clear except at the extreme periphery, where a slight opacity adjoined the recipient cornea. To the present time the graft has remained clear, and vision is 6/15 and Jaeger 3 (fig. 11).

The donor eye had been enucleated, 24 hours earlier, for sarcoma of the ciliary body.

Preoperative observations

From the foregoing cases a number of factors have been noted which influence the course of the graft and give an indication of the probable visual outcome.

Elschnig first pointed out that if the graft were to remain clear, it must be placed in contiguity with a portion of relatively clear cornea; that is, leucoma containing corneal elements. The grafted cornea apparently cannot derive sufficient nutrition from scar tissue to maintain its integrity as a transparent tissue. This finding has been amply borne out in this series by postoperative slitlamp observa-

tion of the transplants. In those cases wherein two or three sides of the graft were contiguous to heavily scarred leucoma, the postoperative edema was more pronounced and vascularization and clouding of the graft began earlier and was more profuse than in the more lightly scarred corneas. This was particularly true in case 13, in which the second transplant was contiguous on two sides to the cornea of the previous graft, and on two sides to dense leucoma. The ingrowing vessels were fine, and none were from the former graft, which was only nebulous. This is the only case wherein transplanting within a transplant was attempted. It is probable that several of these grafts would have been clearer had preliminary penetrating or lamellar keratoplasties been performed to supply the needed corneal elements.

It was often found difficult or impossible to determine the amount of corneal tissue in a leucoma. The degree of visibility of the iris, the slitlamp beam, and retroillumination from the iris all contribute to the estimate. In several cases the leucomas seemed quite dense, yet the grafts remained surprisingly clear. In one case, at least, this was explained by the fact that the scarring, although dense, involved chiefly the outer layers of the cornea.

Some leucomas present large superficial vessels which might be expected either to invade the graft or to bleed postoperatively and thus separate the graft from the cornea of the host. This occurred in one case wherein the vessels were incompletely destroyed before the keratoplasty. Such an eventuality may be prevented by sparking along the course of the vessels with the unipolar or bipolar diathermy current. Should there be a heavy mat of vascular tissue on the surface of the cornea, the epithelium together with the blood vessels may be dissected completely away and reëpithelization allowed to take place.

In several cases it was possible to see the iris through an area of relatively clear cornea, whereas in others the leucoma was so dense throughout that no direct view of the interior could be obtained. In the latter event, transillumination was employed to determine the depth of the anterior chamber, and the size of the pupil and its position in relation to the cornea as well as its reaction to light as the transilluminator was flashed on and off.

The presence of anterior synechiae is an unfavorable factor, indicating a severe process with incalculable damage to intraocular structures. The condition, however, is not incompatible with a clear graft.

A cataract, when determined, calls for further surgery following keratoplasty. In two of these cases cataracts were extracted without complication 11 and 34 months after operation.

Aphakic eyes offer, in general, a poor prognosis because approximation of the wound edge is obstructed by vitreous, or because of the deleterious effect of vitreous in apposition to the healing graft. This condition was present in three cases.

Glaucoma may prove a difficult problem. Increased intraocular pressure during the immediate postoperative period gives rise to corneal edema and consequent poor nutrition of the graft, and invites vascularization. If the increased pressure does not supervene until after healing is complete, the edema of the graft is of less serious import. In the presence of glaucoma, transplantation should be preceded by a filtering operation, and in some cases by an iridectomy as well. Unfortunately, in badly damaged eyes the glaucoma may recur after operation, or atrophia bulbi may ensue.

The function of the retina can only be

conjectured from a determination of the light projection. The projection may be good, the retina may appear normal, and the graft may remain clear, yet the visual result may be poor. This was true in one case of the above group. It would be difficult, however, to refuse operation to a blind person unless light projection were extremely doubtful.

Unfavorable external conditions, such as trichiasis, entropion, symblepharon, or dacryocystitis, can usually be remedied before transplantation. One exception to this was the trachoma of at least 50 years' duration, encountered in case 5.

Donors for these keratoplasties have been limited to eyes enucleated for ocular neoplasms. Neither cadaver nor fetus eyes have been available. Eleven eyes were used for the 16 transplants; two grafts may readily be cut from one cornea with the twin knife and scissors. The eyes were either used immediately after enucleation or were placed in saline solution and kept in the refrigerator at 2 to 6 C.° for varying periods to 30 hours. No difference was noted between the postoperative courses of the grafts transplanted immediately and of those that were preserved.

With two exceptions, the corneas of the donors were normal at the time of enucleation; a high-grade congestive glaucoma had been present for 3 and 10 days in these. The postoperative courses of the glaucomatous transplants were practically identical to those of the normal grafts. It would be interesting to know whether a congestive glaucoma of longer duration, or one superimposed upon a chronic, noncongestive glaucoma would render the cornea unsuitable for transplantation.

In the preliminary work on rabbits, the techniques of partial penetrating keratoplasty as practiced by Elschnig, Filatov, Thomas, and Castroviejo were employed. Twin knife and scissors seemed superior in simplicity and adaptability to the trephine or trephine and scissors. Castroviejo has shown that an incision made with the knife is cleaner than that made with the trephine, and that section with the scissors in a straight line is less jagged than a circular section. In operating upon rabbits with the pupil widely dilated, it was observed that a thin opacity of the anterior capsule of the lens developed postoperatively despite the strictest care to keep the scissors from contact with the lens. This opacity was avoided by constriction of the pupil with eserine prior to operation. Atropine and suprarenin ointments instilled at the termination of the operation rapidly dilate the pupil. In no case has the iris adhered to the graft since the adoption of this procedure.

Operative technique

The technique of Castroviejo has been followed exclusively in this series of cases. For completeness, the procedure is described in detail (fig. 12).

Instillations of pontocain and retrobulbar injection of novocaine are used for anesthesia. Akinesia is obtained by the O'Brien method. The lids are retracted by means of sutures, and the globe is immobilized by a superior-rectus bridle suture and, if necessary, by an inferior-rectus suture as well. A section 4 mm. square is outlined in the cornea with a twin knife. Sutures of fine corneal silk are placed in the cornea approximately 1 mm. from the section all around, producing a Maltese cross effect. To accomplish this, the needle is inserted into the superficial layers and, traversing 2.5 mm. of cornea, is brought out at the upper right corner; inserted at the lower left corner, and brought out at the middle of the lower side; inserted at the middle of the upper side, and brought out at the upper left corner; inserted at the lower right corner and brought out at the mid-

dle of the right side; inserted at the middle of the left side and brought out 2.5 mm. beyond. The single suture is left in large loops and smoothed out of the way during the section. A traction suture is then placed in the square section. Fluorescein is instilled to outline the section better. A keratome incision is made at the upper side of the square at an angle of 20 degrees; the remainder of the square is cut out with special scissors

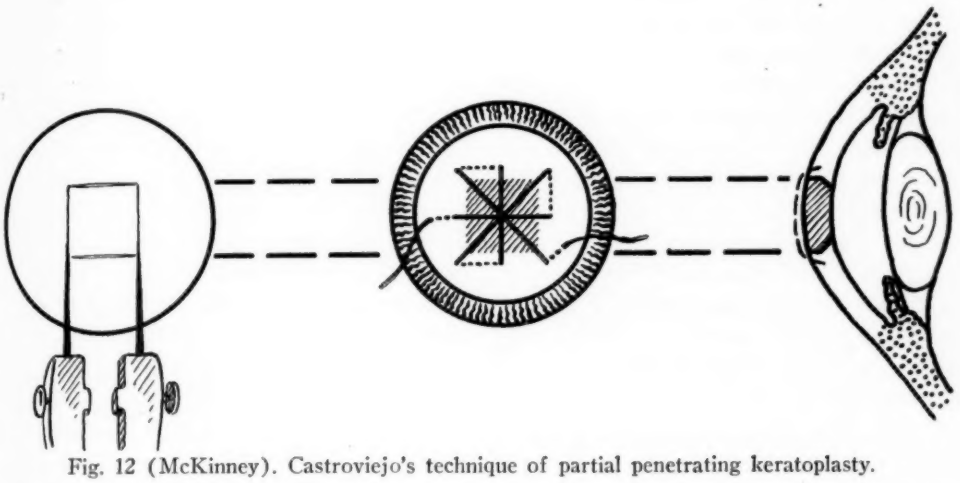

Fig. 12 (McKinney). Castroviejo's technique of partial penetrating keratoplasty.

while traction is exerted with the suture previously inserted. The beveling of the margins to 20 degrees is sufficient to prevent the graft from falling into the anterior chamber. Formerly, the edges of the graft were beveled to 45 degrees, but it was found that this angle made the area of the inner surface too small.

The recipient having been prepared, the donor eye is wrapped in gauze, the cornea remaining exposed, and the graft is cut out in exactly the same manner as the segment from the recipient eye, except that no traction suture is used. The graft is then transferred from the donor to the recipient eye, fitted snugly into the defect, and the suture is drawn taut and tied. If the cornea is thick, the outer portion of the defect will gap slightly. One then has the impulse to tighten the suture to insure close approximation; this, however, produces uneven tension on the graft and cornea and consequent eversion of sections of the graft margin, thus allowing tears to infiltrate and cause excessive swelling of the graft. If the sutures are drawn only moderately taut, the approximation will be completed by the re-formation of the anterior chamber and the postoperative swelling of the graft. The crossed suture and beveling insure maintenance of the position of the graft.

In one case of this group, the needle perforated a thin cornea and the anterior chamber collapsed following the escape of aqueous. The incident necessitated making the initial perforating incision above with a small scalpel, rather than with a keratome. This proved an entirely satisfactory method of entering the anterior chamber, and may be the procedure of choice when the iris is everywhere adherent to the cornea, as the depth of the lens beneath the iris is not known.

Operative findings

In six cases the iris was found to be widely adherent to the cornea. A large portion was removed with the corneal segment, and the remaining adherent por-

tion was freed from the cornea with the spatula, withdrawn, and resected. The ease with which the adherent iris is freed in this manner would seem to obviate the necessity for preliminary traumatizing synechiotomies. In all these cases the iris retracted beyond the wound edge, and in none did it become adherent to the graft.

In two aphakic eyes an excessive quantity of vitreous was lost, making accurate coaptation of the graft with the cornea of the host difficult or impossible. In one of the two, an extracapsular cataract extraction had been performed previously; the posterior capsule was adherent to the cornea and was resected with the segment. This not only caused a loss of vitreous, but also led to the formation of a membrane behind the graft. In a third aphakic eye, no vitreous was lost, although it could be seen in the anterior chamber after the intracapsular extraction, before the cornea became cloudy.

Bleeding was encountered from the deep layers of several of the thicker corneas. This always stopped spontaneously after a time, permitting insertion of the graft into a bloodless defect.

In two of the cases the cornea was dystrophic. The dystrophy of one was of the bullous type; there were epithelial, stromal, and endothelial changes incident to an earlier cataract extraction. The pathologic process had involved all layers of the cornea to some extent. The graft remained clear for several months and thereafter likewise became dystrophic, though to a less extent than the cornea of the host. In the other case, the sections of the excised segment revealed fibrous tissue and lymph spaces at the level of Bowman's membrane and thickening of this membrane; the epithelium, however, was normal. The dystrophy has not affected this graft, which continues to be quite clear.

Mature cataracts were removed through the defect in two cases. The manipulations necessary to removal of the lens were unavoidably traumatizing to the wound edges. In neither case was the graft successful; in both, the anterior chamber remained shallow, and a membrane formed on the posterior surface of the graft.

Postoperative observations

The immediate postoperative course is marked by edema of the graft. The degree of this edema, which is estimated by the brightness of the fundus reflex if the media behind are clear, and by the visibility of the iris beneath, may vary from one which permits a good fundus reflex and good visibility of the iris to one which totally inhibits these manifestations. Clinically, the edema, if continuing beyond the first few days, is an expression of the nutrition of the graft, which depends upon two factors; namely, the apposition of the edges of the cornea of the host and graft, and the condition of the cornea of the host. Castroviejo demonstrated the process of repair in sections taken at intervals after operation. The cleft between the graft and cornea of the host is filled with fibrin, and within a few days epithelium and endothelium bridge the cleft. If the apposition is inaccurate, completion of the latter process is delayed. During the first few days, the graft and cornea imbibe aqueous, but this factor is rapidly eliminated by the growth of epithelium and endothelium when the apposition is good. If, in addition, the condition of the cornea of the host is good, the edema rapidly subsides, and within a few days the graft becomes as clear as normal cornea. If the apposition is not initially accurate, the swelling of the graft aids approximation of the edges; the nutrition is less abundant, however, and the edema does not disappear so rapidly. Nevertheless, the graft will clear if the cornea of the host contains adequate corneal elements. If the

condition of the cornea of the host is poor, the nutrition of the graft will be poor, the edema will persist, and infiltration and vascularization of the stroma of the graft will be observed at the end of the second week. In some cases, the edema seems to have cleared almost entirely and the graft to be progressing nicely when a faint clouding begins, accompanied by a slowly advancing, fine, interstitial vascularization. This process may soon be arrested or may continue from one side of the graft to the other. Apparently, there is a delicate balance between adequate and inadequate nutrition. At any rate, delayed vascularization does not produce heavy clouding and is not incompatible with a fairly clear graft. Whether the infiltration and vascularization begin early or late, the graft tends to become clearer for several months. Nebulae and fine vessels, however, may always remain. The cause of this infiltration and vascularization of the graft is not thoroughly understood. Castroviejo found lymphocytes, polymorphonuclears, plasma cells, and fibroblasts in the graft and immediately surrounding cornea when the transplants were nebulous. He supposed that the vascularization was a defense reaction against infection. In view of the known resistance to infection possessed by ocular wounds wherein nutrition is adequate, however, it would seem more reasonable to suppose that the vascularization is a response to tissue necrosis in the graft as a result of impaired nutrition. In one aphakic eye of this series, wherein there was a profuse loss of apparently normal vitreous and the apposition was known to be poor, the graft underwent almost complete fibrosis without infection of either the graft or the small vitreous bead which presented between the edges of the wound.

There was gaping of sections of the wound in three cases, on the third, sixth, and seventh postoperative days. Each was associated with only slight clouding of the immediately contiguous portion of the graft. The gaps filled slowly and healing was not appreciably altered.

Glaucoma was a complication in six cases, but in only two could the effects of the glaucoma on the graft be distinguished from the effects of poor nutrition. In one of these, acute glaucoma developed 10 months after operation. The lens was removed and the graft regained a normal transparency. In the other, glaucoma was present before keratoplasty and continued afterward. The graft remained intensely edematous, and vesicles, followed by lamellae, appeared on the epithelium. A membrane could be seen behind the transplant soon after the operation.

Infection of the graft occurred in one case; the graft became detached, the defect was filled with scar tissue, and the eye became shrunken. The most likely cause of this infection was the absence of tears as a result of the conjunctival scarring of old trachoma.

Results

The results in this series of keratoplasties are briefly summarized in the accompanying tables:

TABLE 1

CONDITION OF GRAFTS AT LAST OBSERVATION

Graft transparent as normal cornea	3
Graft slightly less transparent than normal cornea	4
Graft nebulous	3
Graft more transparent than original leucoma	1
Graft opaque as original leucoma	3
Graft densely scarred	1
Graft lost	1

Table 1 is a classification of the condition of the grafts after all the healing processes have been completed and all danger of complications has passed. An ultimate opacity of the graft equal to or

greater than that of the original leucoma is considered an unsuccessful transplantation. Any degree of resulting transparency may be expected to give a corresponding improvement in vision, other factors being equal, and is considered a successful transplantation. The visual outcome, however, does not always parallel the transparency of the graft, because of pathologic changes behind the cornea. In the three cases wherein the grafts remained as clear as normal cornea, the vision was 4/60, 6/15, and 6/60 (table 2).

TABLE 2

VISUAL RESULTS

6/60 or better	6
Improved	3
Unimproved	4
Worse	3

The visual results are summarized in table 2. When vision of 6/60 or better was obtained, the result was considered good. In the three cases wherein there was improvement, vision was 2/200, 4/60, and 6/120. In two cases vision was reduced from perception of moving objects to light perception, and in another the graft was lost from infection; these were the three in which vision was classified as worse.

In order to evaluate the visual results fairly, one must take into consideration the pathologic conditions present in the eye receiving the transplant. Leucomatous eyes may be divided into two groups, according to whether they are favorable or unfavorable for keratoplasty. In the favorable eye, the leucoma is not so dense that no corneal elements are present, the anterior chamber is fairly normal, light projection is good, and tension is normal. If only favorable eyes were selected for keratoplasty, the outcome would be successful in a far larger number of cases. The unfavorable eye, however, presents the greater challenge. In this group of cases, 11 of the 16 eyes were regarded as unfavorable. Table 3 presents a comparison of the visual results in the favorable and unfavorable eyes.

TABLE 3

VISUAL RESULTS IN FAVORABLE AND UNFAVORABLE CASES

Favorable cases	5	Successes	80%
Unfavorable cases	11	Successes	55%

COMMENT

The development of successful corneal transplantation has created a fascinating chapter in the progress of ophthalmology. This small series of cases, together with the many others already reported, is encouraging enough to justify the incorporation of keratoplasty in the surgical practice of an increasing number of ophthalmologists.

One may safely predict that in the favorable cases the number and degree of successful results will increase. It is in the unfavorable cases that the accumulated experience of workers in this field will be of especial value, as improved methods are introduced to remedy the numerous complicating factors.

SUMMARY

1. Sixteen cases of corneal transplantation are reported.

2. Preoperative factors which influence the course of the graft and give an indication of the probable visual outcome are recorded, the significance of the amount of leucoma in the cornea of the host being especially stressed.

3. The technique of partial penetrating keratoplasty as developed by Castroviejo,

which was followed in this series of cases, is described.

4. The operative findings and postoperative observations are discussed, the latter with particular reference to nutrition of the graft.

5. A classification and criteria for evaluation of the results are presented.

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RELATION OF CAPILLARY AND CORNEAL OSMOSIS TO GLAUCOMA THERAPY

W. H. Luedde, M.D.
Saint Louis

Glaucoma may be defined as an intraocular edema with inadequate drainage. Edema in any part of the body is directly related to the transfusion or osmosis* of fluid through the walls of the capillary network. Hence, etiological studies of glaucoma necessarily lead to consideration of the causes of edema and the phenomena of the capillary circulation. While Magitot, after extensive research, concluded that all the various forms of the disease, glaucoma, may be included in one category—edema—resulting from an exaggeration of capillary permeability or diminution in the capacity of capillary resorption, it would appear from the experimental studies of Ridley that corneal osmosis may be a factor in the escape of intraocular fluids.

My special interest in the phenomena of capillary circulation began 25 years ago when, in an examination of the corneal limbus with a Czapski binocular corneal microscope, corpuscular movements in conjunctival capillaries were unexpectedly discovered. Inquiry at the departments of physiology of Washington University and of Chicago University indicated that this was a new and untried method for studying the peripheral circulation. Reference to the ophthalmic literature revealed that Schleich had made the same discovery in 1902 and reported it in the Klinische Monatsblätter für Augenheilkunde, only to be obliged to qualify his announcement by a letter published in the following number of that journal acknowledging that priority belonged to Bejardi (Turin) 1892 and 1901, to H. Friedenwald (1888), and that deWecker and Liebreich previously had made the same observation. Apparently the ultimate priority belongs to Coccius, not mentioned by Schleich) who demonstrated the conjunctival circulation microscopically in 1852, one year after Helmholtz made ophthalmoscopy possible.

These repeated rediscoveries indicate that neither ophthalmologists nor internists realized what opportunities were lost when they failed to make microscopic studies of the manifestations of the capillary circulation in the ocular conjunctiva.

Louis Warfield, writing on arteriosclerosis and referring to the capillary circulation 30 years ago, was obliged to make his deductions from observations of the capillary circulation in frogs and cats. He stated that "the farther away from the heart the measurements are made, the more even the flow. In the capillaries it is quite steady, as anyone can prove by direct examination of the web of a frog's foot or the cat's mesentery. The rate in man is estimated to be between 0.5 and 0.9 mm. per second." On the contrary, direct observations of the conjunctival capillaries revealed that the flow of the corpuscles was by no means steady. The rate by direct stop-

* The word "osmosis" did not appear in the first edition of Noah Webster's American Dictionary of the English Language published 100 years ago. The words "osmose," "osmotic," and "osmometer" made their appearance in the "Appendix of new words" in the edition of 1864. The word "osmose," it was explained, was derived from a Greek root meaning "an impulse." Osmose was defined as "The tendency of liquids or gases of different densities, when separated by a porous membrane or partition, to mingle; the less dense, in most cases, flowing into the more dense, and raising the latter, when a liquid, above the level of the former.

watch measurement over a given length of capillary showed that the rate of flow varied from no perceptible movement to 1 mm. per second. In some capillaries the corpuscles moved first in one direction and then the flow was reversed into the opposite direction, without reference to the pulse rate in the absence of any demonstrable cardiac disability. Such capillary regurgitation was quite marked in a patient with aortic valvular insufficiency; but was discovered also in a 25-year-old man suffering from hyperthyroidism (systolic blood pressure 160), and in another patient just after syncope. Some capillaries gave passage to a column of corpuscles, but only occasionally, and remained invisible and apparently empty during the intervals. I was unable at that time to make satisfactory photographs of these phenomena of the conjunctival capillaries, but Ruedemann some 20 years later with improved specially constructed apparatus was able to do so. "The general character of the conjunctival vascular supply has the greatest similarity to the general vascular supply of the body. ... We see here vessels which anastomose freely, which are stretched, shifted, or thrown in folds by ocular movements, which are exposed to the extremes of outside temperature and are affected by local irritation or pressure." Moreover, these tiny vessels are in a transparent membrane, the conjunctiva, and their background is the white sclera so that contrast aids in the discernment of corpuscular movements.

The intensive research of the last decade regarding capillary circulation has fully confirmed our earlier observations of its variability. Capillaries are not merely tiny tubules conveying blood from arterioles to veinlets. Their living wall of endothelial cells constitutes the final barrier between the blood stream and tissue cells. Krog estimated that the area of capillary wall available for fluid interchange in a man whose muscles weigh 50 kgm. (110) would be 6,300 square meters (1.57 acres) and Landis commented that the "total blood plasma volume would be filtered through his 6,300 square meters of capillary surface within 10 seconds at a capillary pressure of 10 mm. Hg if there were no force retaining fluid within the blood capillaries." Vasomotor control normally prevents the simultaneous opening of the entire capillary area, and the colloidal constituents of the blood plasma limit the loss of fluid from the blood stream.

Controversy followed the study of changes in the caliber of capillaries which revealed no actual musculature. One group concluded that variations in the caliber of capillaries were secondary to changes in the caliber of arteries and arterioles. Another group supported the theory that the capillary walls contained contractile elements and that the changes in caliber happened in response to nervous influences. A third group decided that chemical stimuli constitute a major factor in the causation of caliber changes in the capillaries.

Vasoconstriction may be produced by sympathetic stimulation or by adrenalin. A third of a century ago Eliot (quoted by Kuntz: Autonomic nervous system, 1934) suggested that these two means of stimulation were primarily parts of the same process, and that the secretion of adrenalin is a necessary accomplishment of sympathetic impulses. Kuntz concludes that "the cells in the vasoconstrictor centers are stimulated reflexly by afferent impulses from various parts of the body. Thus, vasoconstriction may be elicited by stimulation of the sensory portions of any spinal nerve and certain cranial nerves."

According to Bayliss, vasodilator impulses may be caused by antidromic con-

duction. The first substance liberated at the periphery as a result of antidromic stimulation or local injury, according to Dale, is *histamin,* which directly causes vasodilatation of the minute blood vessels with which it comes in contact. Histamin has been mentioned as a factor in the etiology of glaucoma.

Capillary permeability is presumably greater in dilated capillaries, but Hesselman's experiments indicate that increased permeability does not depend entirely on capillary dilatation. The capillary wall is a nonsecreting membrane permeable to water and crystalloids, but relatively impermeable to plasma proteins. E. M. Landis examined critically so-called changes in capillary permeability in relation to the laws of physical force. By his special direct microcannulation method, Landis was able to make repeated measurements of capillary pressure, and to determine pressure in various parts of the capillary network. He found by calculations and direct observation "that *arteriolar* capillary pressure is higher than *venous* capillary pressure by a significant amount. . . . In man, the arterial capillary pressure amounts on the average to 45 cm. of water" (equal to 22 mm. of mercury), and "venous capillary pressure to about 32 cm. of water" (equal to 16 mm. of mercury).

It will be noted that under average conditions in the general circulation, according to Landis, the arteriolar capillary pressure is just above the average normal intraocular pressure, thus facilitating transudation of fluids into the eye, while the venous capillary pressure is equal or slightly less than normal intraocular pressure as measured by the Schiötz tonometer, permitting absorption. The secretion of fresh humor thus favored under average conditions may cause the escape of aqueous already present through efferent venous channels, but it is apparent that a trivial change in capillary pressure might unbalance this exchange of intraocular fluids and tend to an abnormal retention. This unbalance may promote a general edema behind the iris, hence there is no significant accumulation at the barrier; which Snellen insisted must happen if glaucoma began by retardation of the outflow from the anterior chamber into the canal of Schlemm.

While the origin of intraocular edema must be sought in the extensive capillary network of the uveal tract, opinions differ about the distribution of the transudate from the capillaries. Does the increase of intraocular content implied by hypertension, which must begin as an edema of the uveal tract, remain so; or is more fluid added to the vitreous humor, causing it to swell and compress the choroid? Redslob found that a definite amount of swelling of the vitreous followed a change of only 0.1 in the *p*H. He also reported that a minute addition of fluid (0.02 c.c.) to human vitreous at normal tension caused an elevation corresponding to 60 mm. Hg (Schiötz). Friedenwald estimated the rate of formation of aqueous in dog's eyes as 1.0 cu. mm. per minute; and in a human eye, with secondary glaucoma, as 1.5 cu. mm. per minute. However, actual proof is not available that volumetric increase in the vitreous humor plays a major part in the etiology of glaucoma.

It may be easier to visualize an edematous and vascular increase in the thickness of the choroidal bed, but microscopic sections of old glaucomatous eyes often show marked atrophy rather than swelling of the choroid. In all tests it is well nigh impossible to reproduce and maintain the natural conditions of the living ocular tissues. Hence, diversity of opinions is not disconcerting.

With arteriolar constriction, such as is

produced by epinephrine, peripheral resistance is increased, raising the general blood pressure; but capillary blood pressure is lowered because less blood enters the capillary network. These reactions noted by Landis in the study of the general circulation tend to explain the successful reduction of intraocular pressure secured by Hardesty in the constitutional treatment of glaucoma by epinephrine and ephedrine.

Conversely, Thiel reports that intraocular pressure is elevated after inhalations of amyl nitrite, although blood pressure is lowered by general capillary dilatation. Capillary dilatation within the eyeball might be expected to initiate hypertension. Similarly, it has been reported that certain antipyretics and caffeine, which dilate cerebral vessels, have apparently produced some increase in intraocular pressure. Bonnefon found rapid constriction of the neck of rabbits was followed by a drop in intraocular pressure, presumably by lessening the arterial supply of blood to the head. Slow constriction of the neck created a rise in intraocular pressure, probably by first elevating venous pressure sufficiently to prevent resorption.

Instilled into the conjunctival sac, Landis found that adrenalin or cocaine by arteriolar constriction, diminished conjunctival edema. By direct osmotic absorption, these drugs would have the same effect on edema within the eyeball; but both are mydriatics and their immediate constriction of arterioles, tending to reduce intraocular transudation, is followed by dilatation of the pupil, with tissue compression of veins of the iris and possible obstruction of the iridocorneal angle. This might retard absorption of intraocular fluids sufficiently to increase hypertension by retention of the aqueous. Thus is afforded a possible explanation of the contradictory ocular effects of epinephrine when instilled into the conjunctival sac, as first proposed for glaucoma therapy by Wessely and especially manifested by its stronger solutions, such as Hamburger's glaucosan, and reported likewise by John Green in his observations on suprarenin bitartrate. To follow the epinephrine instillation promptly with a controlling miotic, such as eserine or stronger solutions of pilocarpine—perferably of their alkaloids in castor oil because they seem to act more quickly than ointments, pastes, or aqueous solutions—has been proved effective clinically.

In the general circulation of the body, Landis found that when injury produces local edema, it is due "fundamentally to increased capillary permeability with easy passage of proteins and water through the endothelium." But agents which reduce *local* capillary pressure prevent fluid from collecting, even though the capillary wall is severely damaged. Hence, in contusions of the eyeball, often complicated by secondary glaucoma, the early use of epinephrine, locally, followed by mild miotics (*not* atropine) is a logical and, clinically, a satisfactory treatment.

Arteriolar dilatation, active hyperemia, acute or chronic inflammation, heat, histamin, or anything that increases the capillary flow and elevates capillary pressure, will facilitate filtration of fluid from the capillary network into tissues and has a tendency to cause edema of intraocular tissues. Hence the combination of vasoconstrictors with miotics is logical glaucoma therapy.

Under the ordinary or average conditions, the intracapillary pressure in the venous portion of the capillary network is below the colloid osmotic pressure of the blood. Thus absorption of fluid from the tissues is favored. However, obstruction of a few veins or venules may cause the rise of capillary pressure, prevent

absorption, and thus permit fluids to accumulate in the tissues. Likewise, capillary pressure is relatively high in dependent parts of the body, owing, primarily, to the hydrostatic pressure of the blood in vertically placed veins. These observations of Landis on the mechanical effect of posture and tissue obstruction on venous pressure and permeability indicate a possible, important factor in the diurnal variations in intraocular tension, which Theil demonstrated in healthy eyes but which are more marked in glaucomatous eyes. Rest in bed with low pillows or none may be a factor in the relative increase of intraocular pressure noted in early morning hours. Another factor that must not be overlooked in promoting ocular drainage during the waking hours is the constantly recurring activation of the mechanism of accommodation, including the contraction of the pupil, the forward traction on the vitreous and choroid, and the retraction of the scleral spur at the base of the iris by the contraction of the longitudinal fibers of the ciliary muscle opening the iridocorneal angle.

That decrease in the general colloidal osmotic pressure of the blood may cause glaucoma is graphically illustrated by the frequent occurrence of glaucoma in epidemic dropsy.

The first description of this disease was given by Maynard, who reported 100 cases that occurred from 1908 to 1909. Subsequently, Murkeji reported observations on 253 cases in 1927; Kirwan, to whom ophthalmology will remain indebted for a comprehensive study of the disease, reported 325 patients who came under his care in outbreaks in Bengal and Calcutta during 1929-1930 and 1932-1933. Kirwan closed his first report with the statement: ". . . this subject presents a large field for experimental work, and, if carried out, one can look forward to the day when a great part of the problem of glaucoma will be solved by therapeutic measures or methods of prevention."

In June, 1936, Kirwan reported another outbreak of this interesting disease, 1934-1935, during which period 1,695 cases of primary glaucoma in association with epidemic dropsy came to the Eye Infirmary, Medical College Hospital, Calcutta, under his direction.

Clinically, this "Glaucoma associated with epidemic dropsy is characterized by very high tension occurring in both eyes at the same time, no lowering of the tension by miotics, the presence of a normal or deep anterior chamber, the absence of any inflammations or external signs of congestion, the occurrence of glaucoma in persons irrespective of age, and glaucoma may be the first manifestation of the toxemia."

The toxemia is apparently caused by "the ingestion of diseased, stored, parboiled rice as patients quickly recover after stoppage of rice in early cases; more advanced and late cases take a much longer period to recover according to the duration of the disease. Relapses invariably occur if the rice is resumed too early. These are liable to occur even when healthy rice is eaten, suggesting that infection of the rice occurs in the intestines of the patient.

"In the eye, microscopic examination of the filtration angle shows that the canal of Schlemm and the tissues in the immediate vicinity do not show any abnormalities either in cellularity or increased fibrosis. The epithelium of the ciliary processes also does not show any change. Increased dilatation of the blood tissues is seen in the subepithelial connective tissues of these processes. There is also an enormous dilation of the choroid, but there is no evidence of any inflammatory process to be found in the whole uveal tract."

Kirwan named as primary factors in the production of an increased amount of aqueous humor:

(1) "Decrease in the colloid osmotic pressure of the serum.

(2) "Increase in the permeability of the capillary wall so that the albumen molecules can pass through from the capillaries into the aqueous humor. The albumen molecules are much smaller than those of other proteins in the blood and can pass out more easily than others into the aqueous. The change in surface tension is closely associated with the change in permeability. It is probable that the histaminlike body which has been proved to be present in some cases associated with epidemic dropsy produces similar reactions.

(3) "Increase in hydrostatic pressure in capillaries."

He adds, "It is obvious that the operation of these three factors serves to aid the transudation from the capillaries into the aqueous humor in the eyeball and to hinder reabsorption from the latter thereby causing an increased amount of aqueous humor."

If eating "diseased stored parboiled rice" can produce such changes in the colloid osmotic pressure of the blood plasma that glaucomatous hypertension may be the "first manifestation" of the toxemia, then other toxins may be suspected of similar effects. Kirwan's reports seem to compel the abandonment of the theory that glaucoma is essentially a local ocular disease. Our ophthalmic horizons must expand in regard to this malady. The whole patient must be studied. As a result of such investigation, the possible perfection of prophylactic procedures against glaucoma becomes an eventual probability, while surgery retains an emergency role of great importance.

Kirwan found it necessary to resort to surgical relief (Elliot's trephining operation) in all except the very earliest cases of epidemic dropsy. His recorded failures after simple iridectomy, which were invariably followed by an actual increase of intraocular tension, recall the so-called "Malignant" glaucoma.

Von Graefe estimated that somewhat less than 2 percent of iridectomized eyes were actually made worse by iridectomy. Schmidt, 75 years ago, called attention to the fact that this type of reaction was strangely present in both eyes of the same individual, even though years intervened between the performance of an iridectomy on the two eyes. In such a case, which I reported in 1922, the enucleation of the second eye was avoided by the creation of a filtration scar at the limbus, but four days after the operation a fresh hemorrhage into the anterior chamber brought with it an explosive glaucomatous attack. Instillations of pilocarpine were helpful, but eserine aggravated the condition. The hemorrhages continued at intervals for six weeks. The simultaneous occurrence of intestinal hemorrhages and variation of general systolic blood pressure, from 115 mm. to 195 mm. within 48 hours, indicated the general nature of the fundamental disorder.

Intensive research on osmotic pressure of colloids may some day furnish blood tests by which clinical ophthalmology may anticipate an attack of glaucomatous edema. For the present, close observation of various reactions, local and general, may furnish a clue.

Leiter, by experiments on dogs, showed that chronic hypoproteinemia tends towards an extravascular accumulation of fluid, to the degree of outspoken edema, when the plasma protein was reduced to 2 percent or less; but it was found that

this edema could not be induced without an adequate supply of salt and water. E. M. Landis called attention to the clinical fact that a physical tendency toward edema can be neutralized by restricting the intake of salt and water, or by increasing their excretion through diuretics. Several ophthalmologists, among them the late Dr. A. E. Ewing of Saint Louis, have believed that a restricted salt intake was helpful in chronic glaucoma.

The last three decades have made every ophthalmologist familiar with the ocular significance of focal infections—especially those of the nose, throat, and mouth—in the etiology of ocular diseases. Repeated clinical experiences indicate that they may be an important factor in chronic glaucoma, and in the precipitation of acute attacks.

A lady, aged 63 years, had several acute glaucomatous attacks in one eye which were rather too easily controlled with miotics. Vision was satisfactorily restored and as long as she had no trouble with the other eye she disregarded my advice that possible rhinological foci of infection should be considered before surgical relief, to prevent recurring glaucomatous attacks, might be undertaken with reasonable safety. After a year, sclerosis and incipient opacity in the lens caused impairment of vision to 20/60+ in the affected eye; but vision in the other eye being normal, no urgency for cataract operation existed. Her family physician, who regarded glaucoma and cataract as purely ocular diseases, discouraged rhinological examination and took the patient to an ophthalmic colleague who was willing to operate for cataract without being bothered about possible rhinological contraindications. Enucleation followed three months after cataract extraction. When the patient returned to me four months later, convinced by her disaster that she should have followed my advice in the first instance, vision had begun to fail in her remaining eye. A simple chronic glaucoma was present, hypertension was easily controlled, and vision improved to 20/20 by miotics. The radical treatment of the old infection of both right and left antrum by a rhinologist was followed by further improvement in central vision and by a striking enlargement of the visual field. Reference to other similar experiences must be omitted here.

It is as yet not possible to state whether focal infections appear among the entries in the etiological field of intraocular hypertension through alterations in the blood plasma, by allergic, intraocular swelling, or through low-grade metastatic uveitis.

Last year, certain unexpected clinical results from the use of subcutaneous injections of splenic extract in glaucoma aroused speculations about the possibility of securing a definite control of capillary osmosis through this therapeutic agent. Continued laboratory and clinical investigations revealed an unexplained lack of constancy in the results; and yet its unquestionably favorable effect in a few cases seems to warrant further study to discover, if possible, the reasons for the uncertain action that was discovered both in animal experiments and in its effect on glaucomatous eyes. A simple review of some of the salient points that led to the proposal to use this preparation in glaucoma may clarify the appreciation of our difficulties in arriving at a final evaluation of its usefulness. In any event, the effects it produces seem to pertain to its modification of the osmotic pressure of the blood plasma, so as to favor rapid absorption through the capillary network.

Dr. Thomas Paul, a dermatologist of St. Joseph, Missouri, about 10 years ago noted a report of Moncur in Munich regarding the antiallergic function of the

spleen. The relation of allergy to many forms of eczema is well known. Stimulation of the spleen by X-ray radiation was reported to have had a definitely favorable effect on cases of chronic eczema. Moncur had derived the idea of using this method from the fact that many patients with chronic eczema had an eosinophilia and splenectomized dogs also had a marked eosinophilia. Thus an impaired antiallergic splenic function, it seemed, might be a factor in eosinophilia and chronic eczema. Moncur's clinical experiment was successful to an encouraging degree but obviously could not be followed up indefinitely because unrestrained X-ray radiation of the spleen might have other dangerous consequences.

Moncur's report stirred the clinical imagination of Paul, the dermatologist, and moved him to try original experiments by injecting, subcutaneously, splenic extracts that were prepared at his request by the Armour laboratories. His results were so good that he persisted in its use and was astounded to receive from his patients reports of relief from migraine—asthma—gastric disturbances—dysmenorrhea—and allied conditions, in addition to notable improvement in those cutaneous disorders that were related to allergy and angioneurotic edema. Dr. Paul reached a plausible concept that the migraine that was stopped by the splenic extract was an intracranial edema not unlike the external cutaneous edemas which disappeared after the injection of the splenic extract. (The immediate effect on these noticeable external edemas has been well attested by other clinical experimenters.) From the contemplation of these results, Dr. Paul decided to ask his colleague, Dr. E. A. Miller, in eye, ear, nose, and throat practice, for a chance to try the effect of splenic extract on the intraocular angioneutrotic edema of glaucoma. Dr. Paul supposed that one might get relief from glaucomatous pain, as had happened in the cases of migraine. As Dr. Paul originally phrased it, migraine might be an edema of the brain and glaucoma a migraine of the eye. Their tests were noticeably successful. Drs. Paul and Miller reported this experience to the Missouri State Medical Association in May, 1937.

However, my own use of splenic extract would probably have been deferred indefinitely if it had not been for Dr. M. Goldenburg of Chicago, a wide awake and intrepid ophthalmologist who, nearly a year after Miller and Paul's report, told me he had noted some good results from its use. Thus encouraged, I tried it first of all in one of those desperate situations that sometimes confront us in glaucoma. One of my colleagues, a Saint Louis surgeon, had in his left eye a simple chronic glaucoma which seemed to be well controlled. Under close observation and the regular use of miotics for some months, it was noted that there was no further loss in the visual field; central vision was 20/15 and intraocular tension as measured with the Schiötz tonometer apparently remained within normal limits. The omission of miotics for several days because of a surgical emergency in a distant city, was followed by a severe acute glaucomatous attack. The high intraocular hypertension would not yield to intensive local medication, and central vision was reduced to mere recognition of hand movements at three feet. The eye was stony hard and painful. The cornea was steamy and prevented ophthalmoscopic examination of the fundus. Retrobulbar injection of novocaine solution afforded no relief. General medication with ephedrine failed also. Surgical drainage offered the only hope, but a bacteriological test of the conjunctival sac showed a pure culture of pneumo-

cocci. Under these conditions 2 c.c. of splenic extract (40 percent) was injected and repeated in 24 hours with immediate amelioration of the condition. After three days, vision had returned to 20/15 and intraocular tension became normal. Similar results have been obtained in other cases, but not invariably. In our experience, persons of the general type of pudgy adiposity seemed to get more benefit than others; but the number treated is too small to permit generalization. Experiments on guinea pigs by Dr. John Auer of the Department of Pharmacology, Saint Louis University School of Medicine, and by Dr. Max Ellis, Department of Physiology, Missouri University, on dogs, to test the antiallergic effect of splenic extract, were reported personally to have been inconclusive because of contradictory results. Neither experimenter was able to explain the results which at times appeared to be strikingly positive, but which showed such inconsistency by comparison with controls that no positive answer was possible. Clinical results have similarly lacked consistency. Dr. Ellis attempted to find a reason by preparing the extract himself, but was unable to secure better results in that manner. Armour's extract of spleen, supplied in 40 and 150 percent ampules, appeared to be generally good, but subject to the same uncertainties that had been noted.

It is evident that further biological and chemical studies of this medicament are greatly to be desired. If consistency in the good results which have been noted in some cases of glaucoma following the use of splenic extract could be achieved as a result of such investigation, an important contribution to ophthalmic therapeutics might be recorded. It would also tend to illuminate the obscure phases of the etiology of this disease. Probably the experiences with splenic extract may suggest to our biochemists and pharmacologists other methods of greater efficiency and dependability in influencing capillary osmosis through their effect on the osmotic pressure of the blood plasma. These studies must lead inevitably to consideration of the profound electrical and chemical problems related to the osmotic capacity of the membrane or wall surrounding the protoplasm and nucleus of the living cell. In other words, to the ultimate question which defies all science, "What is life?"

Corneal osmosis

We may pass now to the discussion of the role of corneal osmosis, although we have merely touched upon the possibilities of capillary osmosis in relation to glaucoma.

Whether the intraocular fluid is a dialysate through capillary walls (Duke-Elder, Dieter, and others) or a secretion by the epithelium of the ciliary processes (Seidel and Serr), effective lymph drainage might prevent intraocular edema and hypertension. Drainage from the eyeball is dependent on special structural and mechanical factors. Before giving special attention to corneal osmosis as one of these factors, some general considerations appear pertinent.

Acknowledging that the lymphatics of the orbital cavity are imperfectly known in man, Whitnall states that the lymph from the globe presumably passes out around the anterior ciliary and vorticose veins and the central vein of the retina, and eventually reaches the jugular lymph trunks. Some intraocular fluid is presumably absorbed by the veins. Sondermann believes that intraocular tension—similar to the tissue tension in the glomeruli of the kidney—is higher than ordinary tissue tension throughout the body. In the kidney the flow of the venous blood from the glomeruli is retarded. Sondermann reports that beginning in fetal life, increasing firmness of scleral tissues compressed the venous exits causing the intraocular

veins to become overfilled. The oblique passage of the vorticose veins through the sclera favors valvelike compression of these veins, depending on the degree of intraocular tension. Since intracapillary pressure cannot vary greatly from the adjoining intravenous pressure, compression by the scleral wall must contribute to the increase of intraocular capillary pressure, thus creating a condition that favors transudation of fluid into the tissues rather than absorptions from them. The firmness attained by erectile tissues under venous engorgement is well known, hence the marked elevation of intraocular pressure from scleral compression of penetrating veins occasions no astonishment.

The suggestion that under normal conditions there is little or no flow or movement of intraocular fluids or aqueous humor appears to be untenable when considered in relation to the cinematic photographic demonstrations of v. Pflugk and Machemar of the movements of a fragment of congenital pupillary membrane incited by an active accommodative effort.

The demonstrations made by Hensen and Voelckers 70 years ago, that contraction of the ciliary muscle draws the base of the vitreous (located at the ora serrata by Salzmann) forward and inward, while at the same time, by the insertion of its anterior tendon in the scleral spur at the base of the iris, it opens the entrance to the canal of Schlemm, have never been challenged. They have only been ignored. This mechanism must act almost like a miniature force pump, tending to expel the aqueous into the spaces of Fontana and the canal of Schlemm. Reproduction of this mechanism presumably explains some of the good effects of miotics.

Corneal permeability for mydriatics and miotics is taken for granted, for we instill these drugs daily for their intraocular effect. Perhaps the majority of us have given as little thought to the precise action of transcorneal osmosis as Isaac Newton's fellow citizens did to the fall of an apple to the ground, or as Florentinians did to sunrise and sunset until Galileo contradicted the prevailing opinions.

I am obliged to Eugene R. Lewis for directing my attention to the report of Frederic Ridley of London which first appeared not in ophthalmic journals, but in the British Journal of Experimental Pathology. (Inspection of the volume revealed on its title page a literary warning, not to mistake the whirring of bats in the belfry for the music of the spheres. That quotation seemed somehow to prepare the reader for vigorous discussion with no quarter asked nor given.)

Ridley's paper was based on extensive experimental studies. He submitted evidence that the aqueous humor may constantly drain through the cornea into the subconjunctival tissues or conjunctival sac, chiefly via the filtration angle (the pectinate ligament and canal of Schlemm). The cornea is freely permeable but the sclera is *not*. Because, ordinarily, dependence is placed on the permeability of the cornea inward to secure the effect of mydriatics or miotics instilled into the conjunctival sac, the possibility of permeability being effective outward should also have serious consideration. Citing an experiment showing the resistance of Descement's membrane to the passage of fluid when tied over the end of a glass tube, E. Treacher Collins had stated: ". . . as long as the endothelium lining the membrane is intact, there is no filtration into the substance of the cornea; but, if the endothelium is abraded, then filtration into the cornea takes place and it becomes swollen and opaque."

Ridley found that the permeability of of the cornea is greatest at the periphery, where there may be such modifications

of structure in the walls of the canal of Schlemm that continuous active filtration is possible there while the transparency of the cornea centrally is protected by endothelium. Ridley tried vital staining of the cornea and found that the stain clears from the periphery toward the center; also, that the clearing process was hastened by one third if epithelium and endothelium were removed, but that incision of the substantia propria from the anterior surface had *no* effect on the clearing. Furthermore, Ridley believes that the physical properties of the tears contribute considerably to effective corneal permeability. He reports on analysis of tear fluid made by Dr. W. Hurst Brown, showing the presence of more protein in tears than in aqueous humor and corneal-tissue fluid. Thus, tears have a lower surface tension and exert a small but effective osmotic attraction over the approximate 100 square mm. of corneal surface.

Ridley, experimenting with freshly enucleated pigs' eyes, found an outward transudation capacity for the cornea to be 0.81 c.mm. per minute under 25 mm. Hg of intraocular pressure at a temperature of 30°C. The rate was increased to 1.13 cm. when the artificial intraocular pressure was raised to 50 mm. Hg. These figures were the average of steady readings over a period of one hour in each case but are by no means directly applicable to living human eyes. Experimenting with eyes of etherized rabbits, Ridley found that traces of ammonium chloride began to appear on the surface of the cornea 15 minutes after a 1-percent solution had been introduced into the anterior chamber through the sclera under pressure of 25 mm. Hg. When a 1-percent solution of potassium ferrocyanide was used in the same way and a scratch made in the corneal epithelium from the center to the limbus, to which a drop of 1-percent solution of ferric chloride was applied, the blue color reaction could be detected within 5 mintes at the limbus and within 15 minutes at the center of the cornea. These methods were certainly more precise than those on which Leber based his negative reports, because he failed to see droplets of fluid on the corneal surface. If there is continuous drainage of fluid through the cornea then there must be constant renewal of aqueous humor, and the idea of a normally stagnant aqueous humor must be discarded. Ridley's calculations, based on the Donnan theory of equilibrium between a dialysate and the parent fluid, led to the same conclusion; namely that the aqueous is not stagnant. Finally, Ridley considered the presence of katabolic products resulting from activities of the ciliary muscle. The presence of such products in muscle tissue has been shown to cause increased capillary permeability and dilation followed by transudation of fluid to remove these waste products. The assumption of a normally stagnant aqueous humor appears to be generally irrational as a physiological concept and not justified by known facts.

Later, in the same year, in the same journal, Duke-Elder pointed to the speed with which a methylene-blue solution injected from a reservoir into the anterior chamber of an anesthetized eye, passed into the episcleral veins. These veins were deeply dyed in 1½ minutes. Ridley had used an hour to stain a fresh pig's eye by osmosis of methylene-blue solution from the anterior chamber. Duke-Elder conceded that substances can diffuse from the conjunctival sac into the anterior chamber as established by the work of de Ruiter (1853), v. Graefe (1854), and others, and accepted the proposition that substances introduced into the anterior chamber may diffuse through the cornea entering at the site

of the filtration angle, based on the work of Laquer (1872), Gruber (1894), and Leber (1889-1903); but referred to Parson's warning (1904) that the phenomena of the diffusion of foreign substance need bear no relation to the normal flow of fluid. Duke-Elder adds a truism, "No one denies that changes in the circulation influence intraocular pressure profoundly." He then refers to his demonstration that hypotension can occur with maximal vasodilatation, which is equally true, because, as has been shown, there must be other factors besides general vasodilatation to produce edema of intraocular tissue. It appears reasonable to assume that drainage of intraocular fluid may proceed primarily by episcleral veins, but that does not exclude the possibility of subconjunctival or epibulbar osmotic diffusion through the corneal limbus.

A survey of surgical methods for the relief of glaucoma indicates that corneal permeability as demonstrated by Ridley may be an important factor in the establishment of external filtration. The recent studies of Otto Barkan and his associates seem to indicate that many cases hitherto designated as primary may be classified as secondary glaucoma. In simple chronic glaucoma they found frequently, though not invariably, "pigment dust and granules" permeating the trabeculae over Schlemm's canal. Comparative observations of normal eyes and of glaucomatous eyes have convinced Barkan and his associates that "the amount of pigment deposition as well as its extent relative to the total circumference is closely correlated to the stage of the glaucomatous process, its duration, and so forth." They believe that this is the first stage of many cases of glaucoma and "that the stoppage of the pores of the trabeculae constitutes the immediate and sole cause of the reduced outflow of intraocular fluids"; in other words, "that the pigmentary block is primary and the cause, rather than secondary and the result, of hypertension." Later stages presumably are those in which the angle of the anterior chamber is closed by adhesions between the base of the iris and the cornea, which may manifest acute so-called inflammatory glaucomatous episodes.

The accumulation of pigmented masses or dust in variable amounts surrounding the venous plexus in the canal of Schlemm and their communicating branches, also in the intertrabecular spaces of the pectinate ligament, on Descemet's membrane, and on the anterior surface of the iris, were reported by Gama Pinto more than 30 years ago. He noted their resemblance to pigmented epithelial cells and believed that in certain microscopic sections he could trace the transfer of such exfoliated cells from the pars ciliaris retinae to the anterior chamber and thence to the spaces of Fontana. He referred to Birnbacher and Czermak, who believed that these were white cells impregnated with uveal pigment, and quoted Panas and Rochon-Duvigneaud as being of this opinion. Apparently, it was Koeppe who first attributed great etiological importance to this distribution of pigment especially in so-called "preglaucomatous" eyes where other evidence of glaucoma had not appeared.

Axenfeld (1922) commenting on the inconstancy of the condition described by Koeppe urged delay in accepting the implied significance for glaucoma. Thiel suggested (1931) that while in certain cases the threat of glaucoma cannot be denied, there is no general diagnostic significance in the deposit of pigment débris, which is simply a senile phenomenon to be found similarly in normal eyes (Koeppe-Yoshida).

Thus this discussion continued for

decades, but no one, to paraphrase Mark Twain's remark about the weather, did anything about it until Otto Barkan tried out the effect of direct surgical incision of these pigment-obstructed trabeculae through as large an arc of the circumference as could be reached under gonioscopic control. The success of this operation constitutes primarily an important contribution to the study of the complex etiology of glaucoma. Apparently, it reduces intraocular tension because it provides more direct access of the aqueous to the canal of Schlemm, and also into the more permeable corneal tissue at the limbus, as demonstrated by Ridley's experiments.

Treacher Collins has pointed out that a wound at the corneal limbus, when free from infection and bathed by the aqueous humor, does not cicatrize. This operation —shall we call it goniotrabeculotomy?— belongs to that small select group based on research rather than on empiricism. It might be classed in this respect with the operation for transfixing the iris which Fuchs had applied for the relief of intraocular hypertension in iris bombé, because he had observed that wounds of the iris often were not closed by cicatrization.

Probably the two most popular operations for glaucoma are Elliot's trephining and Lagrange's sclerectomy. Of these the latter uses the better type of incision to avoid hypotony and late infection, but the general opinion seems to be that the Elliot operation affords more reliable permanent relief. Does it make a difference that in the Lagrange operation a portion of impervious sclera is excised while the Elliot trephine button includes more permeable corneal tissue than actual sclera? Are the well-known failures of the trephining operation, when the opening is back in the sclera rather than forward in the corneal limbus, caused entirely by prolapse of ciliary processes; or may the failure to provide improved access for the aqueous humor to the stroma of the cornea be partly to blame? These questions cannot be answered categorically but supply food for thought.

Sixteen years ago I proposed a modified technique for iridectomy in glaucoma in which reliance for a filtering scar was placed on a diversified incision rather than on the excision of any tissue. This operation repeatedly provided excellent enduring filtering cicatrices. The initial keratome incision into the limbus of the cornea, after it had been split as in the Elliot operation, was purposely made small, so that it could be used as a tiny valve to allow the escape of the aqueous, drop by drop, until intraocular tension had been lowered to a degree that a larger opening might be made without undue risk of prolapse or explosive hemorrhage, or other sequelae. Then by scissors incision "wings" were extended laterally from the nasal and temporal edges of the keratomed safety valve. These lateral "wings" were directed somewhat backward and toward the iridocorneal angle to permit a basal iridectomy.

Two years ago, Conrad Berens presented an operative technique very much like this, but with an important addition. Berens clipped off a small portion from the anterior, that is, the corneal, lip of the wound. The special significance of this corneal tissue excision was not impressive, even though it might remove a particle of Descemet's membrane, as suggested by Treacher Collins in the original discussion of my operation. However, after studying Ridley's discussion of corneal permeability in contrast to the impervious density of the sclera, the excision of corneal tissue was added to the previous technique with an astonishing general, circumcorneal subconjunctival postoperative edema, laterally and in-

feriorly as well, under Elliot's thick conjunctival flap. The results in all these cases have been very encouraging.

Berens in a large series of cases has reported equal satisfaction with results. Therefore, to those ophthalmic surgeons who are not satisfied with results of other surgical methods, this combined technique may be confidently recommended.

Résumé

Glaucoma may be caused by general or local conditions which influence capillary permeability or which obstruct drainage from the eyeball. Hence, adequate glaucoma therapy must be local and general.

Capillary permeability may be affected by vasomotor disorders related to nervous or chemical stimuli, posture, or trauma; also by toxic or nutritional alterations of the blood plasma which may modify the intracapillary colloid osmotic pressure of the blood. Variations in capillary blood pressure are more significant for glaucoma than the general blood pressure. Intracapillary pressure may be relatively low when the general systolic pressure is elevated.

Comprehensive general therapy for glaucoma may include the use of sympathico-tonic remedies for vasomotor control, the elimination of focal or general toxins which may modify the constituency of the blood plasma, and such other medication and regulation which a careful general examination may indicate.

Imperfect drainage from the eyeball may be caused by blockade of any normal venous or lymphatic efferent channel. Clinical experiences and experiments support the preëminence that has been assigned to the iridocorneal drainage angle, because it leads to the venous plexus in the canal of Schlemm and secondarily because it somehow permits transudation of introcular fluids through the corneal limbus into the subconjunctival spaces and possibly to the external surface of the cornea. Hence, local medical treatment properly includes ischemics to reduce local congestion and miotics to open the iridocorneal angle. Successful surgical therapy may include the opening of the iridocorneal angle, transfixion or excision of iris tissue, and the establishment of subconjunctival drainage through the corneal limbus. Because of corneal permeability, it is probably more important to excise a small portion of corneal stroma than to cut into the sclera to secure a filtering scar at the limbus.*

Metropolitan Building.

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METASTATIC CARCINOMA OF THE IRIS*

MARY KNIGHT ASBURY, M.D.†
(By invitation)

AND

DERRICK VAIL, M.D.†
Cincinnati, Ohio

The eye is not a common site for secondary malignant newgrowths. Neoplasms, whether primary or secondary, are very rarely seen in the iris. In 1907, when Proctor and Verhoeff[1] reported a case of metastatic carcinoma of the iris, they believed that this was the first case to be recorded in which such a lesion was limited to the iris. In 1938 Sanders[2] collected three additional cases from the literature and described one of his own, making five reported cases. Sanders stated that the iris tumor had been removed in three of the collected cases (1, 2, 3) and had been examined microscopically. He believed that his case was the first in which the intact globe had been examined microscopically, but he had overlooked a report by Hudson and Lister,[3] recorded in the Proceedings of the Royal Society of Medicine for 1924.

At the time Sanders's report appeared we were studying a similar case, and had searched the English literature for the past decade on the subject, finding only the article by Hudson and Lister. If we add our case and that of Hudson and Lister to Sanders's series, the total number of cases recorded in which metastatic intraocular carcinoma involved the iris only is seven.

Several authors[4, 5, 6] have reported instances of multiple metastatic tumors in the uveal tract, one or more occurring in the iris, quite independent of other deposits in the choroid. One of these (Usher[7]) occurred in a man aged 48 years, who complained of loss of vision in the left eye of 10 days' duration. This was explained by the presence of a gray raised area of retinal detachment involving the posterior pole of the eye. In the lower part of the iris a black "bubble," which the patient said had been present for at least a year, had been observed by an ophthalmologist six months before the patient was seen by Usher. The iris lesion transilluminated clearly. The choroidal growth increased in size, and the left eye was enucleated a month after the first examination. The iris lesion had remained practically stationary while under Usher's observation. The patient died five months later. A solid carcinomatous growth, fully described in the microscopic report, was found in the choroid, and at autopsy the primary neoplasm was discovered in the neck and mediastinum. It was believed to have originated in the thyroid gland. The black "bubble" in the iris was found to be a thin-walled cyst, said by the author to be "metastatic," but the microscopic description is not convincing, and the history of slow growth casts further doubt on this, since metastatic carcinoma in the uveal tract invariably grows rapidly.

A number of eyes in which metastatic carcinoma in the ciliary body invaded the iris secondarily have been described. However, Hudson and Lister stated that they had found but 17 recorded cases of metastatic carcinoma of the uveal tract in-

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† From the Holmes Hospital Ophthalmic Laboratory.

volving the iris. In the five years since the publication of their review about six new cases have been added. The number of instances in which the iris is involved in a metastatic lesion of carcinomatous nature is, therefore, very small. Those cases in which such a lesion is found only in the iris and is limited to that tissue are indeed rare.

Of seven such cases to be discussed, the diagnosis in one (4) was clinical only, and in two others (1, 3) the diagnosis was made after iridectomy, but the absence of other lesions elsewhere in the uveal tract was not confirmed. In a fourth case the tumor was likewise removed by iridectomy, but examination of the eye after death revealed no other lesions. In Hudson and Lister's case no section of the primary tumor was available for study, although a kidney containing an encapsulated malignant tumor had been removed two years earlier. The site of the primary tumor in Sanders's case could not be determined since no autopsy was permitted. The fact that our case was the only one in which the primary tumor as well as the affected eye could be studied and compared led us to publish this report.

Case history

A married white woman, aged 49 years, consulted one of us (D. V.) on November 17, 1937, complaining of an inflamed right eye and blurred vision. In August, 1937, her right breast and the axillary lymph nodes had been resected because of malignancy of several months' duration. A day or two before she left the hospital she noticed that her right eye was tender and inflamed. The inflammation grew worse, and a week later her local oculist diagnosed iritis and prescribed atropine. One month later the patient detected a white spot in the iris near the pupil; this slowly increased in size and vision in that eye was blurred.

Ophthalmic examination in November disclosed the following facts: The left eye was entirely normal. The vision in the right eye, with correction, was 20/25. There was moderate ciliary congestion. The corneal epithelium was somewhat edematous. A spongy, yellow-white newgrowth, intimately associated with iris stroma, almost filled the temporal third of the anterior chamber. Its surface was convoluted, like that of brain coral, and flattened to a small extent where it came into contact with the posterior surface of the cornea. Innumerable small, tortuous capillaries in the newgrowth and on the adjacent iris surface lent a pink color to the lesion when viewed with the biomicroscope. The pupil was distorted and displaced nasally. There was an almost complete posterior synechia. The anterior surface of the lens and the posterior surface of the cornea were studded with gelatinous exudate. The floor of the anterior chamber was filled with yellow-white gelatinous and globular débris, consisting undoubtedly of detached fragments of the neoplasm. The intraocular tension measured 65 mm. Hg (Schiötz). The optic-nerve head showed cupping, and the retinal arteries pulsated. The field of vision was normal.

Although the nature of the general condition was hopeless, enucleation was advised in order to prevent pain. This operation was performed the next day under ether anesthesia.

The specimen was fixed in 10-percent formalin. The globe was normal in size and shape. The sclera was slightly bluish and the cornea was clear. The anterior chamber was normal in depth, except on the temporal side, where it was filled with a white, fungous growth. The mass was four millimeters wide and six or seven millimeters long; slightly crescentic in shape, it partly encircled the pupil. At its widest point it extended from the root of the iris to the pupillary margin.

The tumor appeared to be made up of two rounded lobes. The pupil was almost round, about three millimeters in diameter, and the iris was brown. The ciliary region transilluminated clearly.

When the globe was opened, the vitreous chamber appeared to be normal, the lens and retina were in place, and the lesion seemed limited to the anterior segment.

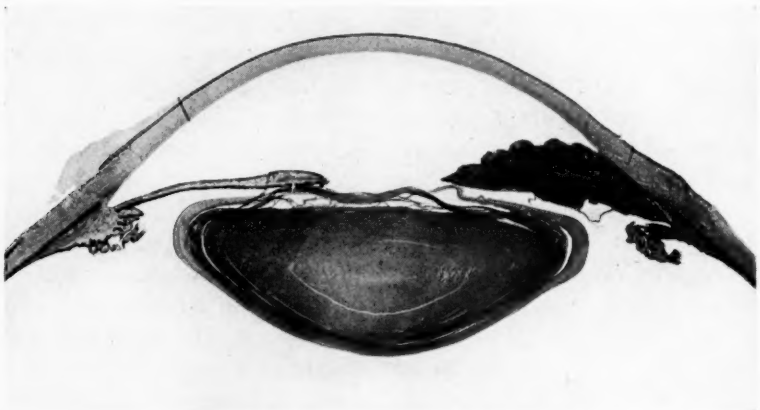

Fig. 1 (Asbury and Vail). Anterior segment. Low magnification. Tumor replacing one limb of iris.

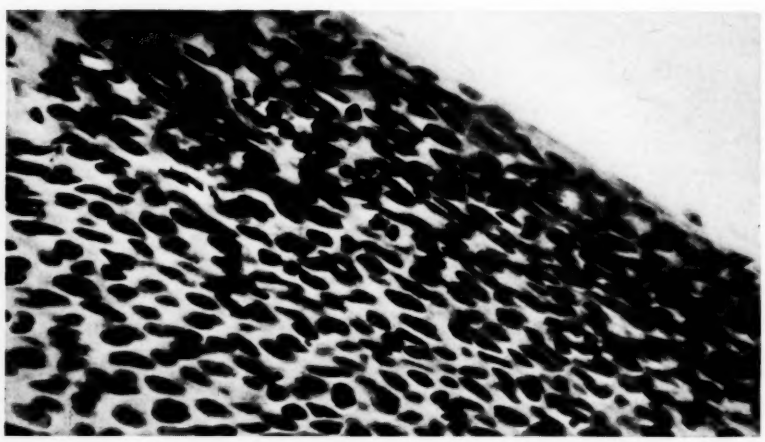

Fig. 2 (Asbury and Vail). Celloidin preparation. Edge of cornea and anterior chamber near angle filled with tumor tissue.

Microscopic examination. In the sections through the pupil and disc one limb of the iris was greatly thickened and replaced by a cellular tumor mass (fig. 1). These were the first sections studied, and a tentative diagnosis of primary tumor of the iris, probably malignant melanoma, was made (fig. 2). However, when the entire series of sections was studied the case assumed a different aspect. A small nest of tumor cells was seen in a pocket on the anterior surface of the iris in the middle third. This increased rapidly in size and extended into the stroma. In the next few sections other isolated (fig. 3) groups of tumor cells were seen in

the stroma and on the surface. In other words, the growth was not well defined, but infiltrating in nature. Within a short distance the neoplasm had replaced the near the center of the lesion, with masses of tumor cells. The neoplasm was not limited by the anterior border of the iris, but had grown into the anterior chamber

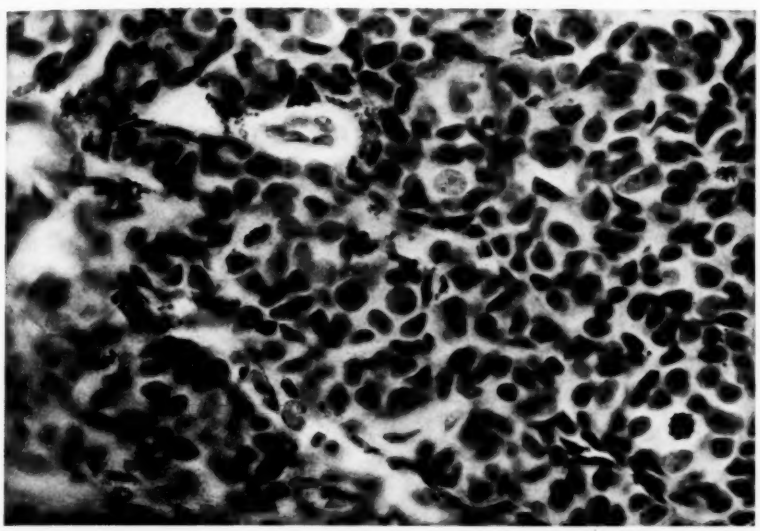

Fig. 3 (Asbury and Vail). Celloidin preparation. Tumor cells in iris stroma.

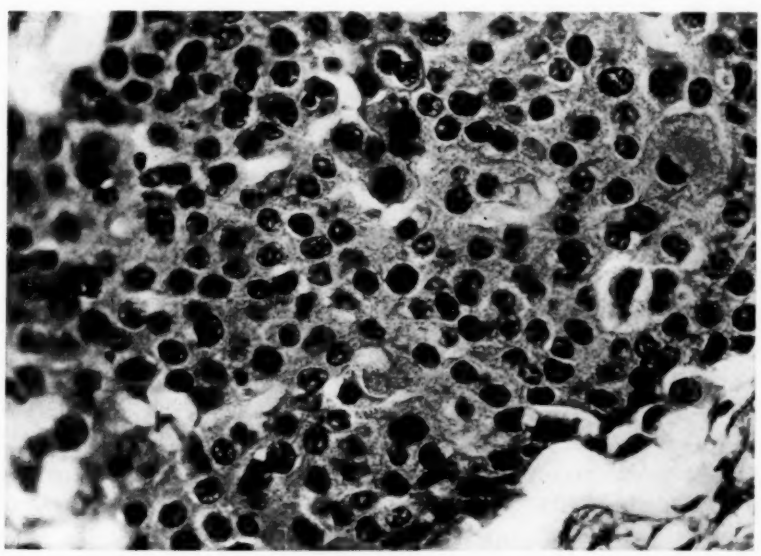

Fig. 4 (Asbury and Vail). Paraffin preparation. Primary tumor in breast.

entire limb of the iris. The posterior pigment epithelium on the affected portion of the iris had been split, and the cysts thus formed were filled with fluid or, and filled the angle for a considerable distance. The tumor was adherent to the cornea, but Descemet's membrane appeared to have impeded its progress in

that direction. The corneal stroma was not invaded.

The entire pupillary border of the iris was adherent to the lens capsule. On the unaffected side a thin fibrous membrane attached to the lens capsule spread over the pupillary border onto the anterior surface of the iris. In this membrane there were several very small groups of tumor cells.

The tumor mass contained melanin, but no pigment was found within the tumor cells; the melanin presumably belonged to the chromatophores of the normal iris stroma which had been engulfed by the tumor.

Within the iris the tumor cells were arranged in lobules of varying size. When stained with silver, a coarse network of fibers surrounded each lobule, but no fine argyrophile fibrils were found in relation to tumor cells. These cells had large round or oval nuclei, which varied considerably in size, and moderate amounts of cytoplasm. Mitotic figures were numerous and very conspicuous. Outside the limits of the iris the tumor cells tended to form larger masses, rather than lobules, and were closely packed; occasional small areas of necrosis were seen. Tumor cells were packed so tightly into the angle of the anterior chamber that they had become spindle shaped (fig. 2). It was this portion of the tumor that resembled most closely primary malignant melanoma. Only near the edges of the growth could the true character of its cells be seen (fig. 3). The neoplasm was confined to the iris and the anterior and posterior chambers. There was an increase in cells in the ciliary body and around Schlemm's canal, but these were inflammatory wandering cells. Sections of the breast tumor were also available for study (fig. 4).

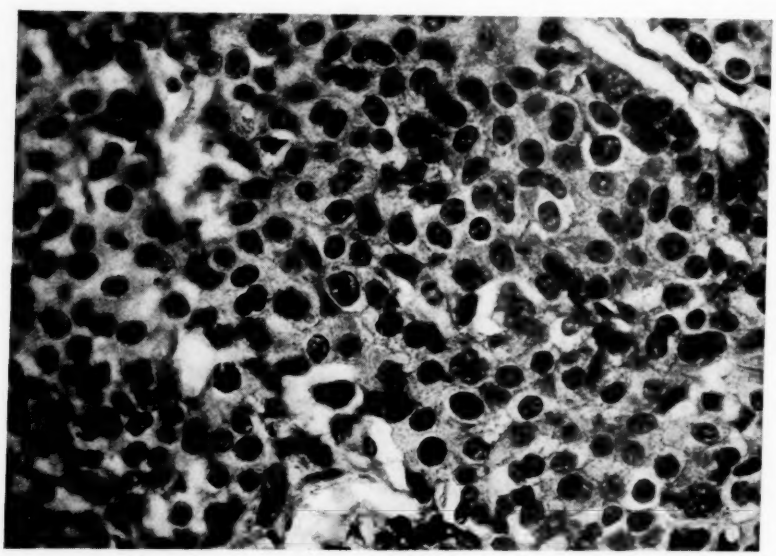

Fig. 5 (Asbury and Vail). Paraffin preparation. Tumor cells in axillary lymph node.

The breast tumor was scirrhous, but nests of cells were discernible in it and in the involved lymph nodes, which resembled very closely the lobules in the iris (fig. 5). Dr. T. L. Terry saw the

sections and concurred in the diagnosis of metastatic carcinoma of the iris, primary in the breast.

The patient died on February 2, 1938, of metastasis to the brain and stomach. There was no autopsy.

Discussion

The time of death is in keeping with the findings of other authors. In practically all the recorded cases of uveal metastases the patient died within a few months after the appearance of the eye lesion. Judging from the illustrations, the gross findings in our case were identical with those of Sanders and Hudson and Lister, except that, as would be expected, the size of the tumors varied. The tumor tissue was confined to the iris or encroached on the anterior chamber; there was no infiltration of ciliary body or cornea. Kreibig[8] reports a case of multiple metastases of a bronchial carcinoma in the uveal tracts. In the right eye a large metastatic nodule in the ciliary body infiltrated the iris, perforated the sclera, formed an extraocular nodule, and also invaded the inner layers of the cornea. This was probably the only case on record, or so the author believed, in which there was infiltration of the cornea.

It has long been recognized that metastatic neoplastic lesions occur much more frequently in the posterior half of the eye than anterior to the equator. Kreibig explains the rarity of metastatic tumors in the anterior uvea on the basis of blood supply. The choroid is supplied by numerous (10 to 20) posterior ciliary arteries, whereas there are only two long ciliary arteries supplying the ciliary body and iris.

Primary carcinoma in the breast accounts for more than 60 percent of the metastatic tumors found in the eye (71.9 percent, Usher). The obvious reasons for the high incidence of mammary carcinoma as the primary focus for uveal-tract metastases are the frequency of primary carcinoma in this location and the nonvital function of the organ, which permits longer duration of the disease and therefore greater opportunity for the dissemination of metastases. In the cases reported in the literature bilateral metastatic lesions in the uveal tracts were frequent. When only one eye was affected, the primary tumor and the uveal metastasis were usually homolateral, as in our case—right breast, right eye.

In 1937, Adda[9] and Kreibig[8] each reported a case of metastatic carcinoma in the uveal tract, and discussed at length the differential diagnosis. In their cases, as in ours, the first eye symptoms were those of iritis or iridocyclitis. Kreibig stresses this point in discussing the differential diagnosis of metastatic neoplasms of the iris. Even though a small nodule was present in the iris when his case was first seen, it was diagnosed as tuberculous nodular iritis. A strongly positive tuberculin reaction, both local and focal, seemed to confirm this diagnosis. The patient was under observation for several months before the correct diagnosis was made. In reviewing the history Kreibig believed that the recurring hemorrhages into the anterior chamber, which almost never occur in nodular iritis, should have led to a suspicion of the correct diagnosis early in the course of the ocular disease.

Metastatic tumors of the iris must be differentiated from primary tumors, from tuberculous lesions, and theoretically from syphilids. In obscure cases of iritis, especially when a malignant tumor is known to be present or has been removed from some other organ, the possibility of metastatic tumor must be considered.

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THE OPTIC PAPILLA IN SEPTIC AND CHRONIC ENDOPHTHALMITIS*

HARVEY D. LAMB, M.D.
Saint Louis

Diseases of the optic nerve can be diagnosed with the ophthalmoscope only as they affect the optic papilla. The optic papilla, that part of the nerve anterior to the cribriform plate, is the only part visible clinically. Little is known as to the anatomic condition in disorders of the optic papilla with the exception of choked disc. There is no definite knowledge of the histology of the commonly observed, so-called toxic optic neuritis or optic papillitis. Henning Rönné[1] of Copenhagen has written the most exhaustive treatise of diseases of the optic papilla and optic nerve that can be presented at the present time. This distinguished authority divides his material into the following categories: 1. Choked disc. 2. Simple or interstitial optic neuritis. 3. Retrobulbar neuritis. It does not seem appropriate to consider all cases of toxic optic papillitis as forms of interstitial optic neuritis. In the well-recognized interstitial type, there is established, as basic, a deposit of bacteria or toxin in the connective-tissue septa that contains blood vessels lying between the bundles of optic-nerve fibers behind the cribriform plate. As clinically observed, a toxic optic papillitis involves equally all parts of the papilla and extends into the adjoining retina. It is improbable that these changes can result from an inflammatory process in a few septa, necessarily lying to one side of the axis of the optic nerve.

In the present paper, the writer does not contend that the inflammatory process in the optic papilla occurring in endophthalmitis can be the only explanation for the clinical findings of toxic optic papillitis. Nevertheless, a study of the reaction of the optic papilla in endophthalmitis affords an opportunity of observing the histologic changes of toxic papillitis and their sequellae.

E. Fuchs[2] introduced the term "septic endophthalmitis" because both in ectogenous and endogenous intraocular septic infection the toxin is deposited principally in the vitreous. From the latter, by chemotactic action, predominantly microphages or pus cells migrate into the vitreous from the ciliary body and retina. In a recent paper the present writer[3] observed that when the intensity of the inflammation is more moderate, macrophages are the prevailing phagocytic cell coming from the ciliary body and retina into the vitreous, and it was suggested that the condition

* From the Department of Ophthalmology, Washington University School of Medicine, and the Oscar Johnson Institute.

should be called chronic endophthalmitis. As in septic endophthalmitis, so also in chronic endophthalmitis the cytologic changes are similar whether the inflammatory process is ectogenous or endogenous in origin. The common cases of so-called chronic uveitis, characterized by precipitates on the posterior surface of the cornea and opacities in the vitreous with or without changes in the intraocular tension and with or without iritis, are really cases of chronic endophthalmitis. The principal clinical changes are due to macrophages, coming as inflammatory exudative cells into the vitreous from chiefly the ciliary body but also from the retina. In the case of a white woman, 25 years old, reported by J. S. Friedenwald and C. M. McKee,[4] a bilateral uveitis and papillitis were observed. Injections of the spinal fluid of this patient into the vitreous of rabbits, dogs, and cats produced a similar inflammatory process. The authors very appropriately designated the condition an endophthalmitis.

In the present study, the optic papilla as part of the lining of the vitreous is under consideration. Because of this relation, it must react together with the ciliary body and retina in septic and chronic endophthalmitis. In other words, the optic papilla must respond to toxins in the vitreous by the giving off from its free inner surface of microphages or pus cells or, on the other hand, of macrophages according as the endophthalmitis is septic or chronic. In a previous paper,[3] it was observed that in septic and chronic endophthalmitis the posterior part of the retina exhibited a greater reaction in the giving off of phagocytic cells than did its anterior part because of the greater blood supply posteriorly. Since the optic papilla has also a good blood supply with numerous capillaries in the glial septa between the optic-nerve bundles, it responds better in endophthalmitis than does the anterior part of the retina, although rarely as well as the posterior part of the retina.

There were investigated for this study of the optic papilla, all eyes with septic and chronic endophthalmitis, whether of ectogenous or of endogenous origin.

Septic endophthalmitis

Tabulation of eyes with septic endophthalmitis is carried out under two categories; namely, those presenting simple septic and those presenting complicated septic endophthalmitis. The differentiation between the two conditions was mentioned in two previous papers (Lamb[3,5]). In the eyes with complicated septic endophthalmitis, there are found associated with large amounts of pus cells in the anterior part of the vitreous, varying degrees of infiltration with small lymphocytes and plasma cells in the ciliary body and choroid. In eyes with simple septic endophthalmitis, the infiltration of the ciliary body and choroid with small round cells is absent. As noted in the listing of total numbers below, complicated septic endophthalmitis is much more common than is simple septic endophthalmitis (44 of the former to 11 of the latter). Therefore far fewer eyes are enucleated early that have a highly fulminant inflammatory process. In the tabulation, it will be noted that all the eyes with simple septic endophthalmitis were removed 15 days and less from the beginning of the inflammation. The retina, optic papilla, ciliary processes, and iris are not infrequently destroyed in simple septic endophthalmitis. In eyes in which the predominant complicated septic endophthalmitis is present, the inflammatory process is either less intensive in degree from the start or, when the enucleation of an eye with simple septic endophthalmitis has been postponed, the toxin in the vitreous becomes weakened with time.

Eyes with septic endophthalmitis now

TABLE I

DATA ON 60 CASES OF ENDOPHTHALMITIS

No. of Case	Patient's Age	Duration of Inflammation	Kind of Penetration	Degree of Perivasculitis	Infiltration within Optic Papilla			Exudate on Surface of Papilla		Connective Tissue on Surface of Papilla	Change behind Cribriform Plate
					edema	pus cells	round cells	pus cells	macrophages		
SIMPLE SEPTIC ECTOGENOUS ENDOPHTHALMITIS											
1290	56	1 day	part. steel in vitreous	papilla entirely destroyed by purulent process				many	few	none	none
1207	13	2 days	corneal wd. from wire	none	mild	none	none	many	few	none	none
1170	51	3 days	part. steel in vitreous	mild	moderate	few	none	many	many	none	mild perivasculitis
1299	74	7 days	cataract extraction	moderate	moderate	none	few	many	many	none	none
2162	50	8 days	corneal wd. unknown f.b.	mild	moderate	none	none	many	many	none	mild perivasculitis
2380	37	15 days	part. steel in vitreous	none	mild	none	few	none	many	incipient	none
240	?	?	?	mild	intense	none	none	many	moderate	none	none
COMPLICATED SEPTIC ECTOGENOUS ENDOPHTHALMITIS											
544	36	2 days	part. steel in vitreous	mild	mild	few	none	moderate	few	none	none
61	12	4 days	corneal wd. from weed	moderate	moderate	few	none	moderate	few	none	none
1428	?	5 days	corneal wd. unknown f.b.	moderate	moderate	few	none	many	moderate	none	none
2168	51	6 days	part. steel in vitreous	mild	moderate	none	none	moderate	moderate	none	mild perivasculitis
999	36	7 days	part. steel in vitreous	mild	mild	none	none	moderate	few	none	none
2119	38	7 days	trephine	papilla entirely destroyed by purulent process				many	few	none	mild perivasculitis
562	24	9 days	corneal wd. screwdriver	mild	moderate	none	none	moderate	moderate	incipient	none
1330	4	2 wks.	wd. limbus from fork	mild	mild	few	none	many	many	incipient	none
2204	23	2 wks.	orbital cellulitis	papilla entirely destroyed by purulent process				few	few	none	mild perivasculitis
2172	27	3 wks.	corneal wd. piecé steel	moderate	intense	none	none	many	many	incipient	none
2273	63	3 wks.	cataract extraction	moderate	moderate	none	few	few	many	none	mild perivasculitis
785	34	3 wks.	cataract extraction	mild	moderate	few	few	few	few	none	none
98	30	3½ wks.	wd. equator from shot	mild	moderate	none	few	many	many	granulation tissue	none
2211	21	3½ wks.	limbus rup. fr. contusion	mild	moderate	none	none	none	many	incipient	mild perivasculitis
1021	25	4½ wks.	corneal wd. from nail	mild	mild	none	few	many	many	granulation tissue	mild perivasculitis
2282	48	4½ wks.	pt. steel in vitreous	mild	moderate	none	none	moderate	moderate	incipient	mild perivasculitis
1041	67	5 wks.	wd. limbus from glass	mild	mild	none	none	few	moderate	none	none
1374	74	5 wks.	corneal wd. piece steel	mild	none	none	none	none	few	none	mild perivasculitis
1970	28	5 wks.	corneal wd. piece steel	mild	moderate	none	none	few	few	none	none
250	20	6 wks.	corneal wd. from wire	moderate	none	none	none	few	moderate	none	mild perivasculitis
482	74	6 wks.	iridectomy for glaucoma	mild	mild	none	none	few	many	none	mild perivasculitis
2218	25	8 wks.	corneal ulcer	intense	intense	none	none	many	many	none	mild perivasculitis
2358	38	9 wks.	corneal wd. unknown f.b.	moderate	none	none	none	few	few	none	none
1899	58	3½ mos.	corneal ulcer	mild	moderate	none	none	few	few	none	mild perivasculitis
1132	42	4 mos.	limbus wd. from shot	mild	moderate	none	none	none	few	incipient	none
1275	4	4 mos.	limbus wd. from glass	mild	mild	none	none	many	many	granulation tissue	mild perivasculitis

1275	4	4 mos.	limbus wd. from glass	mild	mild	none	none	many	few	incipient granulation tissue	none mild perivasculitis

SIMPLE SEPTIC ENDOGENOUS ENDOPHTHALMITIS

1110	55	few days	meningitis staphylococ.	none	mild	none	none	many	moderate	none	mild perivasculitis
1395		6 days	unknown	mild	mild	few	none	many	few	none	mild perivasculitis

COMPLICATED SEPTIC ENDOGENOUS ENDOPHTHALMITIS

1830	48	4 days	corneal ulcer after 20 yrs.	mild	mild	few	none	many	many	none	mild perivasculitis
1534	43	1 wk.	corneal ulcer after 5 yrs.	mild	none	few	none	many	many	none	mild perivasculitis
1163	29	?	corneal ulcer after 10 yrs.	intense	moderate	none	moderate	many	many	incipient	mild perivasculitis
2051	23	?	corneal ulcer after 2½ yrs.	mild	mild	none	none	few	few	incipient	mild perivasculitis
2266	66	2 wks.	abscess on shoulder	mild	mild	few	few	many	many	incipient	mild perivasculitis

40

CHRONIC ECTOGENOUS ENDOPHTHALMITIS

1510	51	2 wks.	corneal wd. from wood	mild	moderate	none	none	few	many	none	mild perivasculitis
2422	21	5½ wks.	corneal wd. from staple	mild	mild	none	none	none	moderate	incipient	mild perivasculitis
545	76	6 wks.	cataract extraction	mild	moderate	none	none	none	many	none	mild perivasculitis
568	36	6 wks.	part. steel in vitreous	none	moderate	none	none	none	few	none	none
2059	14	6 wks.	corneal wd. from rock	mild	mild	none	none	none	few	none	mild perivasculitis
866	30	2 mos.	part. steel in vitreous	mild	mild	none	none	none	few	none	none
64	?	3 mos.	corneal wd. unknown f.b.	moderate	mild	none	none	none	few	none	none
1952	70	3 mos.	trephine operation	mild	mild	none	none	few	moderate	none	mild perivasculitis
2540	82	3 mos.	cataract extraction	none	moderate	none	none	none	few	incipient	mild perivasculitis
1906	47	4 mos.	corneal wd. unknown f.b.	mild	mild	none	none	none	few	incipient	mild perivasculitis
504	59	4 mos.	corneal ulcer	mild	mild	none	none	none	few	none	none
2169	8	?	operations for iris-cyst	mild	moderate	none	none	few	few	none	none

CHRONIC ENDOGENOUS ENDOPHTHALMITIS

1035	76	2 mos.	spontaneous	mild	none	none	none	none	many	advanced	none
1138	45	5 mos.	spontaneous	intensive	intensive	none	none	none	few	none	mild perivasculitis
1430	71	5 mos.	contusion	none	none	none	none	none	none	none	mild perivasculitis
540	62	8 mos.	spontaneous	mild	none	none	none	none	moderate	none	mild perivasculitis
1646	59	9 mos.	spontaneous	mild	none	none	none	none	few	incipient	none
2002	70	2 yrs.	contusion	mild	intensive	none	none	none	none	none	none
1849	27	3 yrs.	contusion	none	mild	none	none	none	few	incipient	mild perivasculitis
2149	77	?	spontaneous	moderate	mild	none	none	none	few	none	mild perivasculitis

20

available in the Ophthalmic Pathological Laboratory collection number 55 and can be classified as follows:

9 eyes with simple septic endophthalmitis of ectogenous origin.
39 eyes with complicated septic endophthalmitis of ectogenous origin.
2 eyes with simple septic endophthalmitis of endogenous origin.
5 eyes with complicated septic endophthalmitis of endogenous origin.

Of these 55 eyes with septic endophthalmitis, there are six in which the optic nerve was incised too far anteriorly to permit a study of the optic papilla, eight eyes with a complete retinal detachment, and one eye in which the inflammatory process was of over six months' standing. These 15 eyes are consequently not included in the tabulation, leaving 40 to be studied.

Four eyes listed under complicated septic endophthalmitis of endogenous origin all show a recent metastatic septic endophthalmitis 2½ years and more after the healing of a perforated corneal ulcer.

Chronic endophthalmitis

Eyes with chronic endophthalmitis in the collection include 14 of ectogenous and 18 of endogenous origin. Among these 32 eyes, the optic nerve was incised too far anteriorly in three, three show complete detachment of the retina, five advanced optic atrophy with glaucomatous cupping, and one advanced optic atrophy without glaucomatous cupping, making 12 eyes with chronic endophthalmitis not included in the tabulation. Twenty eyes with this condition remain in the report.

In the case of each of the 40 eyes with septic endophthalmitis and of the 20 eyes with chronic endophthalmitis tabulated, there is reported the age of the patient, the duration of the endophthalmitis, the degree of perivasculitis of the central retinal blood vessels and their branches in the optic papilla, the relative amount of edema, the relative number of pus cells and macrophages infiltrating the optic papilla together with the relative number of exudative pus cells and macrophages in the vitreous adjacent to the papilla, the presence of connective tissue near the inner free surface of the papilla, and lastly the amount of change (always a perivasculitis) in the trunk of the optic nerve behind the cribriform plate.

The eyes are arranged in the table according to the duration of the inflammatory process or of the endophthalmitis. In the case of all eyes with endophthalmitis of ectogenous origin, this interval can be definitely stated. Where the endophthalmitis is of endogenous cause, this intervening time can be only approximately determined. Septic endophthalmitis, however, is usually so fulminant and painful from its origin, that the patient himself can frequently tell when the inflammation began. In these septic cases, the incipiency can never be more than a few days before the patient consults a physician. Nevertheless, this duration of the endophthalmitis is never set down in the table, unless given in the history by the physician who forwarded the eye.

Findings in tabulated eyes

In the case of three eyes with septic endophthalmitis, the purulent process had completely destroyed the optic papilla.

Perivasculitis of the central retinal stems and their branches in the optic papilla shows in some degree in all but 3 of the 40 eyes with septic endophthalmitis and in all but 4 of the 20 eyes with chronic endophthalmitis. Comparing the eyes with septic endophthalmitis with those with chronic endophthalmitis, it will be seen that there are more eyes with greater degrees of perivasculitis among the septic than among the chronic cases.

The duration of the inflammatory process is found to have no influence upon the degree of perivasculitis. In two eyes with complicated septic ectogenous endophthalmitis of 4 and 5 days' duration, a moderate degree of perivasculitis was present. As pointed out by Lossen,[6] it is a question of the intensity of the inflammatory process. The latter is determined at the time of the introduction of the toxin into the vitreous. The virulence and amount of this toxin in relation to the resistance of the eye define the intensity of the inflammatory reaction. The same facts apply to the papilledema and the exudative cells on the surface of the papilla.

As was found to be the case in the earliest stage of perivasculitis in the retina (Lamb[3]), here also in the optic papilla there was present first only a proliferation of the endothelial cells lining the blood vessels. If the inflammatory process was a little older and the perivasculitis somewhat more pronounced, the cells producing the perivasculitis were mostly macrophages. The proliferated endothelial cells were no longer seen. The derivation of the macrophages from the proliferated endothelial cells is not necessarily to be concluded. If the perivasculitis were still older and more cells were present in and around the walls of the blood vessels of the optic papilla, the macrophages in few to moderate numbers appeared peripherally, and in the walls of the vessels by far the more predominant cells were small lymphocytes and plasma cells. In the optic papilla, both arteries and veins were involved by the perivasculitis to an equal degree.

Between the optic-nerve bundles anterior to the cribriform plate, capillaries are normally present among the neu-

Fig. 1 (Lamb). Case 2218. Section through optic papilla of eye with complicated septic endophthalmitis, showing intensive degree of perivasculitis and papilledema, but no cellular infiltration within papilla; numerous pus cells and macrophages among networks of fibrin as exudate in adjacent vitreous.

Fig. 2 (Lamb). Case 1138. Section through optic papilla of eye with chronic endophthalmitis, showing intensive degree of papilledema and perivasculitis, including perivasculitis of blood vessels in connective-tissue septa behind cribriform plate; few exudative macrophages in physiological cup.

Fig. 3 (Lamb). Case 1906. Section through part of optic papilla of eye with chronic endophthalmitis, showing central retinal artery at right side with irregular perivasculitis, and numerous exudative macrophages in physiological cup at left.

roglial cells composing the thin septa. Where the reaction in the optic papilla was at least of moderate intensity in these eyes with endophthalmitis, there was a perivasculitis around these capillaries. These perivascular cells were comparatively scanty at any one place and consisted of small lymphocytes, plasma cells, and an occasional macrophage.

Papilledema. Edema is the earliest inflammatory change of the optic papilla to be observed in endophthalmitis. Edematous swelling of some degree was present in all but 4 of the 60 eyes with septic endophthalmitis and in all but 4 of the 20 eyes with chronic endophthalmitis. In 19 eyes with septic endophthalmitis and in 7 with chronic endophthalmitis, the papilledema was moderate or intensive in degree.

Cellular infiltration of optic papilla. A study of the tabulation emphasizes the rather scanty cellular infiltration within the optic papilla in endophthalmitis. In 21 of the 40 eyes with septic endophthalmitis, there were practically no pus cells nor round cells (small lymphocytes, plasma cells, macrophages) within the optic papilla with the exception of those participating in the perivasculitis. Fifteen eyes with septic endophthalmitis had a few pus or round cells infiltrating the papilla and one eye with the same condition had a moderate number of round cells in this situation. In none of the 20 eyes with chronic endophthalmitis, in which the intensity of the inflammatory reaction is always less than it is in septic endophthalmitis, were there any infiltrating cells in the papilla.

Fig. 4 (Lamb). Case 2422. Section through exudate just anterior to optic papilla in eye with chronic endophthalmitis, showing macrophages changing to young fibroblasts with their processes.

The optic papilla therefore does not lend itself readily to inflammatory cellular infiltration except of course in tuberculosis. The basic cause for this finding in endophthalmitis must be the particular histologic structure of the papilla. Such cellular infiltration is not uncommonly observed in the retina where endophthalmitis is present (Lamb[3]). In nonspecific inflammations, the brain also does not usually show much cellular infiltration but, as here in the optic papilla, edema and perivasculitis with round cells.

In ordinary clinically observed toxic papillitis, it is probable that cellular infiltration is practically nonexistent. In many cases manifesting the latter, the etiologic toxin from focal infection, and so forth, must be primarily deposited in the vitreous. In this way, the changes would resemble an endophthalmitis although of course in a comparatively mild form.

Fig. 6 (Lamb). Case 1035. Section through optic papilla of eye with chronic endophthalmitis, showing thick layer of connective-tissue membranes on anterior surface of optic nerve.

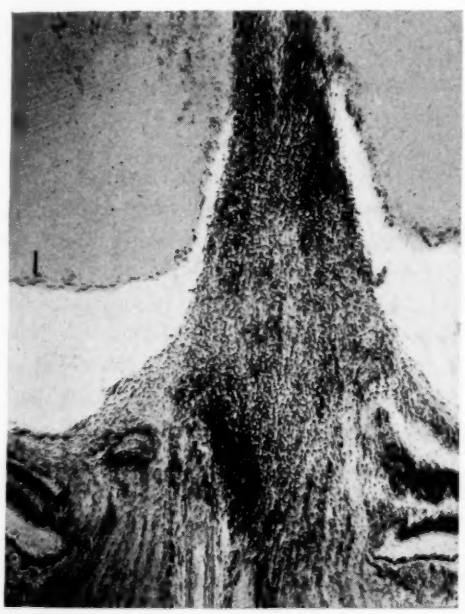

Fig. 5 (Lamb). Case 1275. Section through optic papilla of eye with complicated septic endophthalmitis, showing large conical-shaped mass of granulation tissue extending into the vitreous from the optic nerve.

Exudative cells on the surface of the optic papilla. Next to the perivasculitis, the emigrating phagocytic cells or exudative cells from the inner free surface of the papilla produced the most characteristice change in the reaction of the optic nerve to endophthalmitis. It is exactly the same process as has been described for the ciliary body and retina. Little attention has been given to the manifestations in the optic nerve in cases of endophthalmitis. It is the aggregation of exudative processes in the ciliary body, retina, and optic papilla, bringing about the entrance of pus cells or/and macrophages into the vitreous, that was the essential factor in both acute and chronic endophthalmitis.

In the tabulation, it is noted that all but two eyes with endophthalmitis show at least a few pus cells or macrophages in the vitreous adjacent to the papilla. In the case of these two eyes, that happen to be examples of chronic endogenous

endophthalmitis, macrophages were present along the inner free surface of the ciliary body or the eye could not have been included in the tabulation. It is noteworthy that in all eyes with septic endophthalmitis some macrophages were present in the exudate from the optic papilla. Even when the inflammatory process was but one or two days old, macrophages were observed in the adjacent vitreous. In all but 7 eyes out of the 40 with septic endophthalmitis, the macrophages were comparatively numerous in the exudate. The macrophages in the vitreous near the papilla were, with few exceptions, much less numerous in chronic than in septic endophthalmitis, because the intensity of the inflammatory process was less in the former. The pus cells being very actively migratory, and the macrophages less so, it follows that the pus cells generally accumulate in the vitreous away from the optic nerve, while the macrophages usually assemble close to the papilla.

Formation of connective tissue on the inner surface of the optic papilla. As was stated to be the case for the inner surface of the ciliary body (Lamb[7]) and of the retina (Lamb[8]), macrophages in the vitreous near the papilla may, when the inflammatory process has subsided, become fibroblasts and form connective tissue. This finding was noted in the tabulation in about one third of the eyes with septic endophthalmitis and in about the same proportion of those with chronic endophthalmitis. An incipient stage in the new-formation of connective tissue fibrillae associated with small capillaries to form granulation tissue was present in three eyes with septic endophthalmitis. The transformation of macrophages to fibroblasts was present in one eye with complicated septic endophthalmitis in which the inflammatory process was 9 days old and in another with simple septic endophthalmitis of 15 days' duration. In none of the eyes was connective tissue developing within the papilla.

Connective tissue therefore develops first and principally on the inner free surface of the optic papilla in toxic papillitis just as is the case with the ciliary body and retina. The grayish-white color and indistinct outline of inflammatory or postneuritic optic atrophy is accordingly due to a connective-tissue membrane on the anterior surface of the optic disc.

The last column in the tabulation lists the eyes in which a perivasculitis was present around the small blood vessels in the connective-tissue septa just behind the cribriform plate. Out of 40 eyes with septic endophthalmitis, and out of 20 eyes with chronic endophthalmitis, 22 and 12, respectively, show this finding. The perivasculitis was mild in degree in all but one eye, in which with chronic endophthalmitis a moderate degree was observed. The production of this perivasculitis behind the cribriform plate must be due to the penetration of the toxin from the vitreous to that part of the optic nerve.

Summary

The optic papilla was studied in 40 eyes with septic endophthalmitis and in 20 eyes with chronic endophthalmitis. In the latter, macrophages instead of pus cells were the prevailing phagocytic cells emigrating into the vitreous. Perivasculitis in the papilla was observed in 53 eyes out of the 60 with endophthalmitis. Both the central retinal arteries and veins in the papilla were equally involved in the perivasculitis. Papilledema occurred in 52 eyes, and in 26 it was moderate or intensive in degree. In regard to cellular infiltration within the optic papilla, 15 eyes with septic endophthalmitis presented a few pus cells or round cells. The remaining 25 eyes with septic endophthalmitis and all of the eyes with chronic

endophthalmitis showed practically no inflammatory cells within the papilla. Cellular infiltration is therefore not a characteristic finding in nonspecific toxic papillitis. On the other hand, phagocytic cells migrating into the vitreous from the optic papilla were noted in all but two eyes with endophthalmitis. This exudation of pus cells and macrophages into the vitreous is accordingly characteristic of nonspecific toxic papillitis. In all eyes with septic endophthalmitis, notable numbers of macrophages were present among the pus cells, anterior to the optic disc. In this situation, also, the macrophages were, with few exceptions, much more numerous in septic than in chronic endophthalmitis, because the intensity of the inflammatory process was greater in the septic cases. As a sequel of toxic papillitis, there occurred the transformation of macrophages in the vitreous near the papilla into fibroblasts. Connective tissue, developed from the latter, produced a membrane of varying thickness on the inner surface of the optic disc. The ophthalmoscopic appearance of inflammatory or postneuritic atrophy is therefore due to this connective-tissue membrane lying on the inner surface of the optic papilla.

All sections and microphotographs used in this study were prepared at the Laboratory for Ophthalmic Pathology, Washington University School of Medicine.

1230 Missouri Theater Building.

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THE PATHOGENESIS OF RETINITIS PIGMENTOSA (SCLEROSIS PIGMENTOSA CHORIORETINALIS)

II. Local symtomatology

Lizzie Levy-Wolff, M.D.
Tel-Aviv, Palestine

I. 1. Having recognized that in retinitis pigmentosa the primary cause is the affection of the vessels, we at first turn our attention to the intraocular blood supply. Two different systems are to be distinguished: (a) the capillary plexus of the choroid and (b) the retinal vessels, which are end-arteries and represent the ramifications of the arteria centralis retinae. In their course they are accompanied by the homonymous veins. There are two different basic types of the fundus oculi both of which we must regard as physiological. One form is found principally in the fair-haired type of man whose skin shows an even and strong pigmentation after exposure to the sun's rays. Generally, in this group of men, the pigment epithelium, separating choroid and retina, is evenly and well developed. Therefore, upon examination with the ophthalmoscope a field of projection is seen of nearly even tint, light red to medium red, in which the vessels of the retina and macula stand out plastically. The second system of vessels, the choriocapillaris, remains invisible. The second form is the so-called tesselated fundus; it is frequently found in the dark-complexioned types or in individuals whose skin does not show an even distribution of pigment and which reacts on exposure to the sun or quartz rays with brown spots of pigment on a lighter, pigmentless basis, the so-called freckles; in this form both vascular systems can be easily distinguished in the fundus oculi. The dark-red and engorged vessels of the choriocapillaris are seen to form a much-ramified plexus and to stand out clearly from the gray-black intervascular spaces. Above them pass the retinal arteries and their concomitant veins; the macula in this form is not conspicuous and often becomes visible only through reduced illumination in the erect image. The ground for these phenomena lies in a defective development of the pigment epithelium. When the light falls on it, the pigment membrane appears transparent and the vascular system of the choriocapillaris underneath becomes visible. Between the two basic forms there are, of course, many transitional stages. The well-known image of the fundus in albinism is only one of the numerous varieties.

In all of the 22 cases of retinitis pigmentosa which I have examined with this in mind, I found the choriocapillaris to be visible in some places, and in 100 percent of my examinations I could convince myself of the fact that this vascular system was very seriously damaged. The visible vessels proved to be changed by sclerosis; they stood out grayish-yellow from the darker intervascular spaces. The retinal arteries were always contracted. In the early stage the veins were of normal caliber. In the retinal arteries the morphological changes were never so marked as those I regularly perceived in the choriocapillaris in the very first stage. As the disease progressed the retinal vessels also contracted, so that in the typical cases the differences of caliber between the arteries and the veins gradually disappeared. In these late stages paired vessels were seen, fine as a hair, and frequently only orientation by means of the optic nerve would disclose whether you were dealing with an

artery or a vein. Unfortunately I had to dispense with exact measurings of the caliber as the necessary apparatus were not at my disposal, but the contraction of the vessels is so striking that their evaluation by figures is unnecessary.

Looking back, it does seem very strange that these facts accessible to every examiner should have remained entirely unnoted. It can be explained only by the fact that the ophthalmologists up to this time have adhered to the fiction that so-called retinitis pigmentosa is an affection of the retina of obscure origin. Now and then there is a hint, however, that the choroid may "have a share" in it, but nowhere in the literature, so far as I can see, is it pointed out that just the choroid with its choriocapillaris is the site and point of issue of the local morphological changes. We are even more justified in saying that the retinal vessels also become affected in the course of the disease.

Here well-known illustrations from the classic textbooks show the whole choriocapillaris, as far as it is visible, greatly changed by sclerosis. The retinal vessels, although apparently narrow, are not nearly so seriously affected. One text adds the note: "In advanced cases the choroidal vessels also may be calcified."

2. The condition of the optic nerve is early recognized and has been frequently described in the literature. The disc has a peculiar waxlike appearance; that is, the reddish color has nearly faded out and is replaced by a soft yellowish-orange tint. Whereas in the early literature, in general, we still find the term "atrophia nervi optici," the opinion today prevails almost without dissent that the waxlike discoloration is the manifestation of an ischemic process.

The anatomic conditions of the optic disc deserve a brief review: The optic fibers on entering the globe find specially favorable conditions for their nutrition. For this reason the nerve fibers in the posterior optic pole continue to function for a comparatively long period, in spite of the grave ischemia in retinitis pigmentosa. The optic nerve is not a nerve in the usual sense of the word but an evagination of the brain. Like the brain it is sheathed by the three membranes of the brain; namely, dura mater, arachnoidea, and pia mater. Between these sheathes are the intervaginal spaces separated from each other and ending in the globe as in a blind alley. At their blind end they absorb the entire lymph supply of the retinal interstices. Before gathering at the optic disc, the lymph again intensively irrigates all prepapillary fibers. The proper nutrition of the optic nerve is undertaken by branches of the ophthalmic artery dividing within the intervaginal spaces. The central vessels enter the optic nerve from behind, about 15 to 20 mm. retrobulbarly, and pass in its axis up to the disc. In crossing the lamina cribrosa the central artery has its only communication with the system of the ciliary arteries. Its ramifications function as real end arteries.

Thus the nerve fibers upon entering the globe are again refreshed by the lymph which abundantly irrigates them here and by means of a new accession of blood from the central retinal artery. The papillo-macular bundle deserves special consideration. Twelve millimeters before it enters the globe, it moves bodily to one side from its hitherto central position and passes into the lower lateral part of the cross section of the optic nerve. This bundle has the shape of a wedge with its base at the periphery of the optic nerve. Favored by its anatomical site, the fasciculus papillo-macularis derives great profit from the fresh blood supply of the central retinal artery, besides being refreshed by the returning lymph of the retinal layers.

For a better understanding of the syndrome of retinitis pigmentosa we should take into account that the central retinal artery is much less affected by the disease of the vessels than is the system of the ciliary arteries forming the choriocapillaris. Hence undoubtedly the papillomacular bundle, as to its nutrition, is in a comparatively more favorable position than are the rest of the nerve fibers.

3. The pigment is the peculiar characteristic of the disease. It has a distinctly deep, dark color and appears in the fundus shortly after the first objective symptoms of the disease. The deposit, as a rule, begins in the intermediate zone, in the neighborhood of the equator of the bulbus oculi. The forms of the pigment particles are diverse and not characteristic. In part they follow the course of the vessels, partly they are also scattered without reference to them. At first they are so scanty and sparse that they often are discovered only after a long search. Somewhat later the typical bone-corpuscles appear in the periphery and thence they slowly progress to the center. But so far as I could observe in the cases I have examined they for the most part avoid the center itself and the parts around it. Here deposits of a noncharacteristic type are predominant.

All these facts have long been known, but never has an explanation been advanced why retinitis pigmentosa especially favors the equatorial and peripheral zones for the deposit of its pigment. Without conceiving of retinitis pigmentosa as a manifestation of a vascular disease, here and there attention has been drawn to the fact that the capillary system of the retina had been injured first in the periphery. This is quite true; but an explanation of this favoring of the equatorial zone has been lacking.

In one of the foregoing sections, dealing with the blood vessels, it was established that the choriocapillaris is far more affected than the system of the retinal vessels. Here I wish to quote from Krückmann in his chapter on the uvea in Axenfeld's well-known textbook. Dealing with the distribution of the vessels in the choroid he writes: "The areas of the arborizations of the arteriolae are largest in the equatorial zone. Thus just at the equator there are frequently areas of the choriocapillaris in which the blood circulation is comparatively slow and where at times, under pathological conditions, it soon comes to a standstill. An aggravating factor is that the arterial anastomoses are comparatively scanty in the equatorial zone, so that here a sufficient supply can no longer be brought by means of arterial but often only by capillary tributaries. On the other hand, arterial anastomoses in the macular area are plentiful as also in the anterior portion of the choroid. In these places, if need be, a very quick compensation is made possible, should one of the arteriolae fail."

These anatomical facts finally supply the long-awaited solution! The secondary local morbid process, as has been shown, does not take place in the retina but chiefly in the choroid. As a rule, the retinal vessels participate at a later period. The pigment is deposited in the retina exactly at the projection planes corresponding to the diseased portion of the choroidal choriocapillaris; namely at the equatorial zone. Later on, when the capillary system of the retina becomes affected, the so-called bone corpuscles are formed. Slowly the process advances from the periphery to the center. The shape of the bone corpuscles is a replica of the smallest capillary dendritions of the retinal system.

II. 1. Before turning now to the anatomic specimen, a few anatomic points should be considered: The retina is divided into two separate portions, the

cerebral portion and the neuroepithelial portion; that is, the true sensory neuroepithelium. Of the three neurons of the retina, one belongs to the sensory neuroepithelium, whereas the two other neurons are part of the cerebral portion. The dividing line of these two functionally different parts passes within the outer plexiform layer and not, as supposed before, on the border of the outer plexiform and outer nuclear layers, because within the outer plexiform layer the external processes of the bipolar cells of the inner nuclear layer are interwoven with the inner processes of the cells of the outer nuclear layer forming a close interlacing. This division is expressly indicated by the vascular supply. Whereas the first neuron, that is, the layer of the sensory neuroepithelium, is quite without blood vessels and is nourished only by the lymph flowing to it in a system of lymph interstices, the brain portions of the retina, that is, the second and third neurons, have a distinct vascular and capillary system at their disposal, the arborizations of the retinal end arteries. The visible marks of this capillary net can be easily traced up to the outer border of the inner nuclear layer (Eisler), but this field of supply doubtless comprises also part of the outer plexiform layer (Leber). The pigment membrane has a special place. Ontologically part of the retina, it is separated from it after full development. The pigment epithelium adheres so closely to the choroid that, in cases of detachment of the retina, it remains attached to the internal layers of the choroid, thus forming a unit with its adopted tissue. Whether the pigment membrane gets its nourishment also from the choriocapillaris or, like the neurosensory epithelium, is supplied by the lymph flow has not been established, so far as I know. It is very probable that both systems share in the task of nourishing the pigment membrane and, in case of emergency, act vicariously.

2. This fact is corroborated by the latest researches of Gasteiger on the vascular changes in retinitis angiospastica. This process involves mainly the arterioles and capillaries of the retinal end arteries. The choriocapillaris in most cases is only participating. The increased blood pressure in the syndrome of the pale hypertony of Volhard, producing a very hypertrophic muscular wall of the vessel, apparently prevents the intima from proliferating. Gasteiger in the fundamental study of his cases paid special attention to the pigment membrane which, through Koyanagi's theories, had become the center of the process. His researches showed that the pigment epithelium was either not changed at all or had comparatively slight changes, even in severe disorders of the circulation, the consequences of which could be seen even in the layers of the neurosensory epithelium nourished by lymph. From these facts it is evident that the pigment membrane is not dependent at all on the supply from the lymphatic system alone, but that even a diminished blood supply from the more or less damaged choriocapillaris proves sufficient to keep the pigment epithelium active and efficient. This result seems strange to a certain extent, as it was the general opinion that the pigment epithelium is especially vulnerable. Gasteiger could show that it is much more resistant than all the other layers of the ontologically related retina!

Furthermore, interesting and very remarkable is the fact that in Gasteiger's specimens the pigment cells which had immigrated into the retina were not derived from the pigment membrane, but that in all these cases there were leucocytic elements originating from the choroid, pigmented and affected by steatosis, which had traversed the lamina vitrea

and the intact pigment epithelium. With Gasteiger, I emphatically wish to call attention to the results of Schieck who emphasized the fact that pigmentations of the retina are due to diseases of the choriocapillaris.

3. Turning now to the microscopic changes underlying retinitis pigmentosa the facts may be considered in the light of our new knowledge. Unfortunately, I have just now no specimens of my own and therefore must rely on my former notes and on publications of other authors from the literature. In a recent paper by Asayama and Takagi from the Ophthalmological Clinic at the University of Kioto, "concerning the appearance of lipoids in the pigment epithelium in retinitis pigmentosa," there are four reproductions in black and white of sections of the retina, three of which give a good view of the changes in the entire retina and the adjacent choroid. This is the eyeball of a 56-year-old man which was trephined because of glaucoma simplex two years before enucleation. The ophthalmoscopic findings then showed, besides a glaucomatous excavation in this eye, the typical syndrome of retinitis pigmentosa. In both eyes: "a pale disc, contracted vessels, and the retina darkened and studded with black spots similar to bone corpuscles." The fact that the choroid was atrophic, its vessels calcified, and partly even entirely obliterated was expressly stated by the authors but not taken into consideration and evaluated.

In figures 1 and 2 of their article we see at once a horizontal dividing line clearly passing central to the faintly visible outer nuclear layer; that is, in the outer plexiform layer. The tissues beyond this dividing line, namely, the inner nuclear layer, the inner plexiform layer, ganglion-cell layer, and nerve-fiber layer, are transformed into one structureless mass. Only the external limiting membrane seems to be intact. Within this tissue which has undergone such severe changes, some sporadic lumina of arteries stand out, having a highly proliferated intima. Since the illustrations are not color reproductions, they unfortunately do not allow distinctions of details to be made in the muscular part and the adventitia. The retinal layers on this side of the dividing line, although severely injured, still permit identification of their proper structure. The pigment membrane, under stronger magnification, appears slightly corroded but its continuity is nearly preserved. The tear in figure 1 is undoubtedly an artifact. Thus here also the pigment membrane is the least injured layer of the whole retina. Only the misconception that the pigment in retinitis pigmentosa must originate from the pigment epithelium has caused us to overlook these facts or, if perceived, not to appreciate their real value. Of the choroid there is not much to be found in these sections. The choriocapillaris seems to be nearly destroyed. The pigmented elements are much increased. Now and then lumina of vessels are seen to be more or less obliterated.

4. Thus far we made the mistake of viewing the facts from only one side, from the details, and have failed to grasp the larger connotations. Starting from the fixed premise that the pigmentation phenomena could be causally connected only with the retinal pigment epithelium, we closed the door to further fruitful research and arrived at an impasse. Now having ascertained the vasomotor disorder with its consequences as causal factor of the local process the problem presents an entirely new aspect. The retina is seen to be nourished by three different systems: the retinal capillary system, the lymph, and the choriocapillaris, which participates in the nourishment of the pigment membrane. All these systems,

single or combined, can be involved in circulatory disorders. The pigment epithelium has the best conditions for nutrition, for it is supplied both by the choriocapillaris and the lymph flow. Both systems replace each other vicariously: Thus in retinitis angiospastica—anatomically a circulatory disorder in the capillary area of the retinal arteries and secondarily affecting the lymphatic system to a considerable degree—the comparatively uninjured choriocapillaris takes over the whole supply. The result is: *the pigment layer remains intact.* On the other hand, in retinitis pigmentosa, which is a vascular affection, when the choriocapillaris is most involved, nutrition by the lymph flow suffices for the maintenance of the pigment membrane. Thus the retinal pigment epithelium not only has the best conditions of supply but proves to be still more resistant than the rest of the tissue layers of the retina and the choroid.

The pigment of the retina itself in retinitis pigmentosa has its origin for the most part in the choroid and, together with leucocytic elements and wandering cells, migrates through the membrana vitrea and the intact pigment epithelium into the retina. The pigment membrane itself has only an insignificant share in the process of pigmentation.

Jonas S. Friedenwald and Eugene Chan discovered that in animal experimentation, the transportation of pigment artificially brought into the vitreous humor is carried out by monocytic elements and with the aid of Müller's fibers, which grow hypertropic and assume phagocytic properties. Though the pigment reaches the retina from the side of the vitreous, the ganglion cells of the retina are scarcely injured, but the authors found lesions of the sensory epithelium. Friedenwald does not ascribe these changes to the presence of the pigment introduced, which in itself was borne without reaction, but to the activity of the phagocytic elements. These facts are in accord with my considerations, for, to my mind, in the syndrome of retinitis pigmentosa the degenerative phenomena in the ganglion cells are secondarily produced by circulatory disorders in the retinal end system. Therefore in the rabbit's eye—where the vessels were normal—the retinal ganglion cells were not affected even by the presence of free pigment.

As to the vascular process in retinitis pigmentosa itself, the proliferation of the intima is dominant. It is supposedly due to the combination of two factors: (a) vascular spasm and (b) hypotony. To this end the cholesterin in the blood stream may be regarded as the mediating principle.

The steatosis, for the first time mentioned by the Japanese authors, must be regarded as the manifestation of a *disorder in the general fat metabolism* shown by cholesterinemia which, postulated here for the first time, can always be demonstrated in the course of the disease.

After all these discussions it may be justified definitely to discard the hitherto existing misleading term of the syndrome. In its place I should like to propose a name that is informing both as to the nature of the morbid process and its localization, pigmented chorioretinal sclerosis or *sclerosis pigmentosa chorioretinalis.*

5. The optic nerve, as hereinbefore pointed out, is not a real nerve but an evagination of the brain, and the nerve-fiber layer of the retina represents the extension of its end fibers over a wide surface. It is therefore not surprising to find in the brain changes corresponding to those in the retina. The ganglion cells of the retina correspond exactly to those in the brain. Now it has been proved that the ganglion cells are extraordinarily sensitive to any form of disturbance in the

circulation. Anemia lasting only a quarter hour will bring about changes in the ganglion cells which, however, as a rule, are reversible (Ernst). Inasmuch as in sclerosis pigmentosa chorioretinalis vascular changes in the arterioles of the retina are an early symptom and the slowly falling blood pressure is not likely to remove the anemic condition, the ganglion cells are affected in an early stage. Very soon the characteristic phenomena in the retinal ganglion cells are produced. Yet it may be safely assumed that in a process actuated within the brain itself the eye is not the sole responding organ. I should like to predict with certainty that the *ganglion cells of the brain* will also be found changed in the same characteristic way, when researches are undertaken to this effect. The evidence from anatomical specimens hitherto studied is scanty and insufficient as these connections have never been looked for thus far. The retinal end arteries have their equivalent in the arterioles of the cerebral vessels, which are also end arteries.

This degeneration of the retinal ganglia, only a secondary condition, was hitherto erroneously looked upon as a primary degenerative phenomenon of unknown cause and the actuating process was supposed to be here. Though, for want of material, we cannot, for the time being, show proof that the brain more or less participates in the same process that is also at the bottom of sclerosis pigmentosa chorioretinalis, yet for a long time we have known of two syndromes that show the same process of degenerating ganglion cells, both in the eye and the brain; namely, the two forms of amaurotic idiocy: the Tay-Sachs type and the juvenile form of Spielmeyer-Vogt. Concerning both syndromes, much anatomical material in the literature has been studied. Numerous are the cases in which attempted clinical differentiation between sclerosis pigmentosa chorioretinalis and amaurotic idiocy of the type Spielmeyer-Vogt, has given rise to considerable difficulties. I have examined the literature for such "border-line cases." The diagnostic decisions have always seemed to me very arbitrary and sometimes dependent on contingencies. The absence of bone corpuscles in no way means absence of sclerosis pigmentosa chorioretinalis. As the choriocapillaris is always the first to be affected, the pigment set free through the choroidal process at first is deposited in the equatorial zone only, often as amorphous masses or in the form of minute streaks. The transparent retina covered with pigment dust on the much-calcified choroid as a base, often assumes a slate-gray color with greenish tints, which induced the authors to call such cases atypical. The typical bone corpuscles, as a rule, follow later, when the process has reached the retinal capillary system also.

It is in this stage of deposition of amorphous pigment particles in the equatorial and adjacent zones that the cases of juvenile amaurotic idiocy are brought to the doctor on account of the psychic condition. In this juvenile form of amaurotic idiocy the cerebral symptoms are most striking, at a time when the ocular symptoms are still but little advanced. To my mind, there are differences in degree only, not in principle, between the juvenile form of idiocy and sclerosis pigmentosa chorioretinalis, if the ocular affections are on both sides and symmetrical. In both syndromes, we unquestionably see the same morbid process at work. It appears, of course, that, if the disease begins at an early age, the central nervous system is much more affected than is the organ of sight. On the other hand, psychic changes can be demonstrated in nearly all cases of typical sclerosis pigmentosa chorioretinalis but they

are, as a rule, less marked and less conspicuous than are the ocular symptoms.

The syndrome of the Tay-Sachs type is different. Although the anatomic specimen is nearly identical with that of the juvenile form, the ocular process must be considered separately. The characteristic phenomena in the macula and the lack of pigmentation have nothing in common with the syndrome of sclerosis pigmentosa chorioretinalis. Werncke, not long ago, reviewed the anatomic results in eyes of children who had died suffering from Tay-Sachs disease. His fundamental studies produced very remarkable results. Apart from the typically changed ganglion cells, in 100 percent of the cases the outer plexiform layer, and within it Henle's fiber layer, were most affected. This is just the layer where the retinal capillary system ends. The destruction is brought about by severe edema penetrating down to the macula. Here the liquid masses seem to damage the choroid below by pressure, because at the border of the edema deposits of amorphous pigment are mostly seen, doubtless originating from the injury to the choroid at this place. Choroid and choriocapillaris are not taken into consideration in this paper. Yet, since no further phenomena of pigment are present, considerable changes probably were not to be found.

As to the etiology, Werncke does not venture an opinion. Since he paid no attention to the different vascular systems of the retina he missed the fact that in all his cases there was a vascular disorder which affected solely the retinal vascular system. Only those layers of tissue were destroyed which are supplied by the capillary system of the end arteries; namely, all the cell layers from Henle's layer to the center. Among all the ganglion cells, according to Werncke, there was often not one intact cell.

Thus the two forms of amaurotic idiocy are to be entirely separated from one another, although both syndromes are doubtless based on circulatory disorders of a central origin. In by far the greatest part of the cases until now diagnosed as "a juvenile form of amaurotic idiocy" we are dealing with a variety of sclerosis pigmentosa chorioretinalis in which the cerebral symptoms outgrow the optic phenomena. The Tay-Sachs type of syndrome therefore remains as the sole form of amaurotic idiocy.

III. 1. A symptom often mentioned in connection with sclerosis pigmentosa chorioretinalis is hemeralopia. Whether by accident or not, there was not a single patient among the cases I examined who spontaneously complained of it. After special questions to this effect one or the other vaguely stated that at night he could not see so well as other people. In all of the early cases that I accidentally discovered while examining for some other reason, hemeralopia was not admitted. Therefore it does not seem to be an early symptom. Neither did Axenfeld's case, reported in 1909, nor the two cases of Scheerer show night blindness. Most recent researches announce a connection between hemeralopia and vitamin A, which in considerable quantities was found in the retina (Yudkin).

2. The reduction of vision proceeds parallel with the advance of the disease. As is well known, central vision is the last to disappear. So long as there remain vestiges of peripheral-field vision there is often binocular excentric fixation, but, as a rule, each eye returns separately to central fixation.

3. The field of vision deserves consideration; it has as yet been unduly neglected. At the end of the last century von Gräfe and Gonin recognized ring scotomata as early symptoms of sclerosis pigmentosa chorioretinalis. In 1909 Axenfeld published a case of "rudimentary

pigment degeneration," and for the first time stressed the symmetry of the field of vision. But, as late as in 1927, Scheerer stressed the regularity of this symmetry. Hepburn, in 1908, had described double rings, the inner ring of which was open below in one eye, and above in the other eye. Wilbrand and Saenger also found similar symptoms. In this kind of visual fields the symmetry is not recognized at first sight, but, since I was fortunate enough to discover some such forms among my cases, I was also able to establish the regularity of the symmetry: both rings in themselves are likewise formed regularly, but their symmetrical axes are perpendicular to each other. Wilbrand and Saenger have given themselves to a thorough study of the origin of ring scotomata. A definite point of derivation could not be ascertained, but the phenomenon always originated from heteronymous points. Scheerer appropriately termed these regularly found symptoms, heteronymous ring scotoma, a term which had appealed to me also before I knew of his paper.

It is not common knowledge that bitemporal hemianopsia, less frequently though none the less regularly, may develop from heteronymous scotomata situated laterally or paracentrally. Wilbrand and Saenger expressly stated this in their comprehensive paper. By going only one step further we shall recognize a variety of bitemporal hemianopsia in the heteronymous ring scotoma that always begins laterally. This knowledge certainly could have been gathered much earlier but for the erroneous teaching that in bitemporal hemianopsia the dividing line has to run directly through the center, as in supposed contradistinction to lesions of the optic tract in which the "macula is exempted."

Yet these statements are incorrect and do not correspond to reality by any means. As to whether the macula is affected or not, there is no difference in principle between optic tract and chiasma. The reason for the eventual integrity of the papillo-macular fibers, however, is solely to be found in their central situation while traversing the optic tract, the chiasma, and also two thirds of the optic nerve. But two things must be kept strictly apart: (a) If there is a complete cutting through of the fibers of the tract or a corresponding direct cut of the middle parts of the chiasma, as, for example, by gunshot wounds or fracture of the bone, the dividing line will go right through the center both in homonymous and heteronymous hemianopsia. The fibers of the macula are also cut; thus in the field of vision the centers are affected. (b) But if there is a slowly progressing destructive process (tumor, hemorrhage, meningitis, enlarged third ventricle, and so forth) the pressure, according to physical laws, chiefly affects the site of the pressure and its contralateral pole, which means that the external fibers are subjected to a far greater pressure than is the center. In these cases the macular fibers, situated in the center, are spared in the optic tract and in the chiasma, and consequently the macula is exempted in the field of vision. If at first we find an "exemption" and then later the center of the field of vision becomes affected, we must consider that there may be a destructive process, growing by infiltration, which in the beginning showed only symptoms of pressure but later on, through its infiltrating growth, has brought about a real lesion of the fibers. It is in this respect that the state of the papillo-macular bundle has a certain significance in differential diagnosis.

Several attempts have been made to explain the "exemption of the macula" by its alleged bilateral supply from the cortex cerebri. Yet the researches of Brouwer and also of Poljak absolutely contradict this theory. In unilateral foci of disease in the human brain, both in-

vestigators found the retrograde degeneration of the corpora geniculata externa at only one side. The same result was produced by unilateral operative interferences in the monkey. On the other hand, it is supposed today that every retinal unit is connected with a whole area of elements in the cerebral cortex and, relying on the researches of Best, Goldstein, Monakow, and Minkowski, the cortical representation of the macula is believed to be in the region of the horizontal stripe deep in the calcarine fissure. The possibility of a double cortical representation of the macula, therefore, has been entirely eliminated as the explanation of heteronymous ring scotomata. The problem is mentioned here only for the sake of completeness.

In order to make the relations between heteronymous ring scotomata and bitemporal hemianopsia clearer, it is necessary to deal in detail with the principle of heteronymous lateral hemianopsia and its origin. A defect of both temporal halves of the field of vision produced by the same process, is conceivable only by assuming a process injuring the central decussated fibers of the chiasma by pressure. Conditioned by the arrangement in the interlacing of the decussating fibers, every pressure from without on the medial portions of the chiasma necessarily brings about heteronymous defects of the field of vision situated laterally. Thence, reversing the sentence, it follows that any symmetrical effect on the lateral halves of the field of vision cannot be produced except by a process injuring the area of the medial portions of the chiasma by pressure. With the advance of the pressure-causing process, sooner or later, the undecussated lateral fibers will inevitably become involved; in other words, the bitemporal defect now encroaches also upon the adjacent nasal halves. Finally, the cones of the scotoma, growing from above and below, will fuse and form a binocular ring scotoma, but in most cases the ring will not be completed, especially since in malignant tumors the patient dies before this consummation. Whether the center is also attacked or remains "exempt" is not important in principle, only to the prognosis. By no means does the bitemporal hemianopsia need to be completed before the scotoma invades the nasal halves of the field of vision. The form of the pressure-exerting process and the intensity of the pressure are decisive for an eventual premature invasion by the scotoma on the nasal halves of the field of vision.

The nasal portions of the scotoma are not necessarily symmetrical. If the pressure-exerting agent spreads equally to both sides, symmetrical defects are formed in the nasal halves of the field of vision. But if the process advances principally in only one direction, the undecussated fibers of one globe, are of course, injured more severely than the corresponding fibers of the other eye. Consequently, the pictures in the region of the nasal halves of the field of vision are unsymmetrical.

From these considerations it is evident that bitemporal hemianopsia has no invariable and regular appearance such as homonymous hemianopsia has, in which the mid line is never passed. It is rather the manifestation of a dynamic process on the way to developing heteronymous scotoma to the full picture of ring scotoma. The exact halving of the field of vision with or without exemption of the macula is nothing else than an ideal stage for the justification of the term, incorrect in itself: bitemporal hemianopsia. In reality this postulated ideal is hardly ever found. It would be much more correct to call it a laterally initiated heteronymous anopsia.

There remains only to clear up the question of the exact symmetry in both halves of the field of vision that is regu-

larly observed in all heteronymous ring scotomata of sclerosis pigmentosa chorioretinalis. All heteronymous ring scotomata which I have had the opportunity of following up, while still incomplete, were open in their lower lateral aspect. All scotomata developed symmetrically, starting from the area of the blind spot, then passing to the upper temporal, the upper nasal, and the lower nasal portion where, in the lower temporal sector, both end portions approached or fused, respectively. In the chiasma, according to the investigations of Wilbrand and Saenger, the fibers for the upper halves of the field of vision are situated dorsally, those for the lower halves ventrally. As the chiasma is freely movable and even by pressure from any side does not lie on a bony base, direct pressure has a far greater effect than counterpressure; that is, the order of the defects of the field of vision corresponds directly to the direction of the pressure exerted. By transferring the dynamics of the scotomata to the chiasma itself, the pressure passes symmetrically on both sides from the dorsal medial to the dorsal lateral side, then round the chiasma from above downward, then to the ventral lateral side, finally to fade towards the ventral medial side. As pressure-exerting factors may be regarded tumors, inflammatory processes, and vascular changes. But none of these three causes can be expected to grow invariably so regularly as to produce always the same annular form with its opening towards the lower lateral side. Yet it is conceivable that the pressure-causing principle does not exert the direct pressure itself but avails itself of an always identical and symmetrically formed structure which produces the pressure in its place. Upon looking for such an auxiliary factor we found the circle of Willis. There are numerous well-known cases—Erdmann and Hirsch have often pointed them out—of tumor of the pedicle of the pituitary gland in which the arterial ring closes tightly around the chiasma and causes deep grooves, especially in the dorsal and medial parts of the chiasma. Towards the ventral side the pedicle is inserted between the chiasma and the vascular ring as a pad; which fact has not yet been considered at all, so far as I am aware (fig. 2).

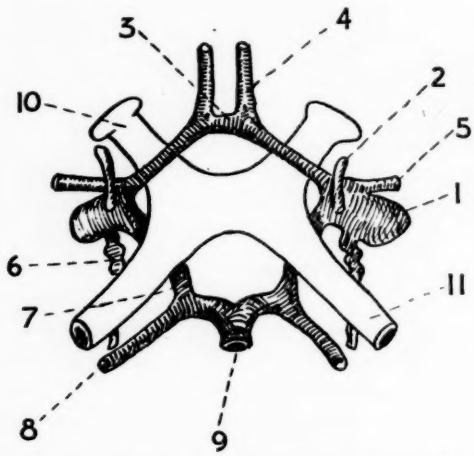

Fig. 2 (Brouwer). Reproduced from "Die Symptomatologie der Erkrankungen des optischen Systems," in Handbuch der Neurologie, volume 6. 1, A. carotis interna; 2, A. ophthalmica; 3, A. communicans ant.; 4, A. cerebri ant.; 5, A. cerebri media; 6, A. chorioidea; 7, A. communicans post.; 8, A. cerebri post.; 9, A. basilaris; 10, N. opticus; 11, Tract. opt.

Thus the chiasma is surrounded above by the carotid arteries and the anterior communicating arteries. Laterally at the curvature, it is situated directly in the angles formed by the carotid and communicating posterior arteries. These latter arteries give way towards the lateral and posterior side so that the pressure on the medial lower portions of the chiasma (affecting the external lower temporal quadrant of the field of vision) is reduced in these places, especially as the infundibulum here acts as a pad between the chiasma and the vascular ring.

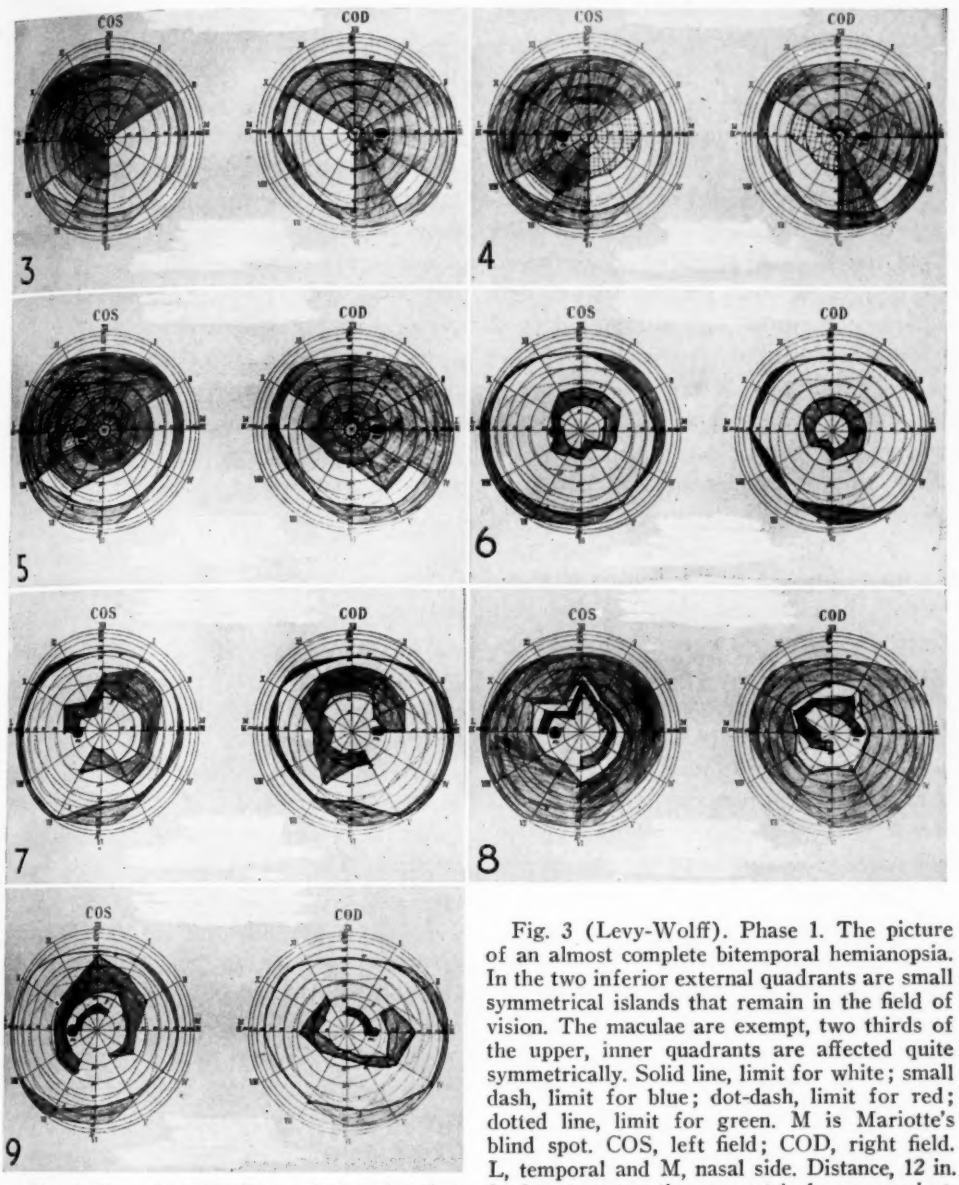

Fig. 3 (Levy-Wolff). Phase 1. The picture of an almost complete bitemporal hemianopsia. In the two inferior external quadrants are small symmetrical islands that remain in the field of vision. The maculae are exempt, two thirds of the upper, inner quadrants are affected quite symmetrically. Solid line, limit for white; small dash, limit for blue; dot-dash, limit for red; dotted line, limit for green. M is Mariotte's blind spot. COS, left field; COD, right field. L, temporal and M, nasal side. Distance, 12 in.

Fig. 4 (Levy-Wolff). Phase 2. Starting from the intact center, the symmetrical cones projecting to the nasal halves of the field of vision are marked by crossbars, dotted lines join the nasal remainders of the field of vision to the temporal ones.

Fig. 5 (Levy-Wolff). Phase 3. In the last stage, the field of vision being fully developed, there is found scarcely a trace of its derivation from the bitemporal hemianopsia.

Fig. 6 (Levy-Wolff). Closed heteronymous ring scotoma with intact periphery.

Fig. 7 (Levy-Wolff). Heteronymous annular scotomata not as yet closed, with intact periphery.

Fig. 8 (Levy-Wolff). Heteronymous ring scotoma still open, with concentric contraction of the peripheral field of vision.

Fig. 9 (Levy-Wolff). Genuine double rings, symmetrical axes perpendicular to one another. The axis of the inner rings is vertical, that of the outer rings horizontal.

These conditions, therefore, correspond exactly to the phenomenon of the heteronymous annular scotomata. Themselves, therefore, they prove to be a classic form of the chiasmal syndrome thus far unrecognized.

As to the diagnosis, theoretically space-narrowing processes at the chiasma itself or at the infundibulum could be taken into account, such as genuine tumors and inflammatory processes; also an enlarged recessus of the third ventricle could be supposed, but it is by far the most probable assumption to think of exactly the same vascular process in the circle of Willis leading to sclerosis, which also in the choriocapillaris actuated the phenomena in the choroid. Heteronymous ring scotomata are the proof already demonstrated clinically that the brain is also affected in the syndrome of sclerosis pigmentosa chorioretinalis.

Still obscure is the problem of the alleged relations between the circular pigmentations of the fundus and ring scotoma. Whereas in former papers the identity of both phenomena was postulated, well-founded doubts as to the correctness of these views were soon published. Axenfeld found well-developed annular scotomata at a time when there was scarcely a trace of pigment deposits. Scheerer, on the other hand, described cases in which pigmentation far overstepped the region of the scotoma. In spite of thorough investigations to this end I could not find any connection myself. Owing to the considerations stated above, we should be in a position to give an unequivocal answer to these questions: There are no mutual relations at all between pigmentation and heteronymous ring scotomata. The pigmentation in the equatorial zone was recognized as the consequence of a local vascular process in the choriocapillaris, whereas the phenomena in the field of vision are the result of a process in the chiasma opticum. Neither process has a direct relation to the other; both are parallel symptoms of an overruling general principle.

With the aid of a field of vision which I have mapped out I should like to demonstrate the development of heteronymous annular scotoma from bitemporal hemianopsia. The patient was 39 years old with typical symptoms of sclerosis pigmentosa chorioretinalis (figs. 3, 4, 5).

Figures 6, 7, 8, and 9 show a number of characteristic types of fields of vision which, although changing in form, I have found again and again, in my cases of sclerosis pigmentosa chorioretinalis.

Summary of part II

Critical study of the local symptoms has led to the following new results:

1. Sclerosis pigmentosa chorioretinalis is the manifestation of a circulatory disorder actuated by the central nervous system.

2. The site of the circulatory disorder in the first place is the choriocapillaris. Later the retinal end-system also becomes affected. The lymphatic system is only passively injured, if at all.

3. The brain portion of the retina represents a complete entity as to its function, anatomy, and supply of nourishment.

4. The optic disc and the papillo-macular fibers normally have the very best nutritional conditions since they profit from both the lymphatic system and the freshly supplied bloodstream of the central retinal artery. Upon entering the eyeball both are "freshened up" again, which is of special importance for the severe circulatory disorder in this syndrome.

5. The pigment deposited in the retina has its origin for the most part in the choroid and is carried into the retina through leukocytic elements and wandering cells. The pigment membrane of the retina partakes only to a very limited

extent in the pigment phenomenon. Owing to its favorable supply, it is comparatively the best-preserved layer and is preserved throughout nearly its whole extent.

6. The pigment favors two definite zones: (a) The equatorial zone, which is the place of least resistance of the choriocapillaris (Krückmann). Here the pigment is deposited as dust or minute streaks; the retina is only the plane of projection on which the diseased parts of the choroid (choriocapillaris) are projected as pigment dust. (b) The periphery: here the pigmentation assumes the form of bone corpuscles; the process slowly advances to the center; by some authors the form is explained to be the cast of the smallest capillary systems.

7. The vascular process in the main is shown by the proliferation of the intima. The cholesterin in the bloodstream apparently is actively involved in the process by being deposited in the tissues injured through ischemia. Vascular contraction and hypotony combined favor this development.

8. The steatosis of the retinal tissue, as described by Japanese authors for the first time, is secondary and also dependent on the percentage of cholesterin in the blood.

9. The degeneration of the retinal ganglion cells is due to the continuous disorder of nutrition.

10. In sclerosis pigmentosa chorioretinalis the ganglion cells of the brain are undoubtedly subject to a degenerative process corresponding to that of the retinal ganglion cells due to the circulatory disorder. Histological studies to this effect in sclerosis pigmentosa chorioretinalis have not been undertaken thus far, but clinically all typical forms show more or less psychic alterations.

11. The juvenile form of amaurotic idiocy, if pigmentation and heteronymous annular scotomata are present, should be considered as pertaining to sclerosis pigmentosa chorioretinalis. In the early form the brain symptoms are prevalent, in the late form the ocular symptoms predominate.

12. Amaurotic idiocy of the Tay-Sachs type is due to a vascular disorder of a different kind, hence it must be strictly differentiated from the Spielmeyer-Vogt type.

13. Hemeralopia cannot be demonstrated regularly; it is by no means an early symptom.

14. Heteronymous ring scotomata are the purest manifestations of a chiasmal syndrome. The absolute symmetry of *both* halves of the field of vision proves that the site of the injuring factor is the region of the circle of Willis.

15. Pigmentation and ring scotomata have no direct mutual relations. Both are parallel phenomena of the same overruling principle.

Epicrisis

The new definition of the syndrome of sclerosis pigmentosa chorioretinalis necessitates also a new order of the symptoms according to their importance: The syndrome is composed of two complexes of symptoms, local and general.

The principal *local* symptoms are: (1) heteronymous ring scotomata = chiasmal syndrome; (2) pigment phenomena; (3) deafness or deaf-muteness, respectively (only in 10 percent of all cases).

Heteronymous ring scotomata are the classic early symptom, which should never be missed; they are the *conditio sine qua non*. Pigmentations of the retina may be valued positively in the diagnosis as a symptom of sclerosis pigmentosa chorioretinalis only if at the same time heteronymous ring scotomata can be found. The three local symptoms can be

easily explained from the respective sclerotic vascular changes.

On the other hand, the *general* symptoms, that is, cholesterinemia, lowered temperature, and hypotony cannot be understood except through the involvement of the diencephalon. Eventual disturbances of the pituitary gland are of a secondary nature and probably due to obstacles to the passage of its secretions. The interruption takes place in the tuber cinereum off the circle of Willis. These processes may occur already in intrauterine life and then lead to malformations such as polydactylia, atresia ani, and other conditions.

Syndromes until now regarded as independent, such as the Laurence-Moon-Biedl syndrome and amaurotic idiocy of the Spielmeyer-Vogt type now turn out to be varieties of the more comprehensive complete syndrome of sclerosis pigmentosa chorioretinalis.

The insulation of the region involved—brain and the connected organs of sense—is nothing extraordinary and conforms to the generally recognized theories of Pal. Owing to the small extent of the affected vasomotor area, the resistance produced in the peripheral circulation cannot give rise to a secondary hypertony and to hypertrophy of the heart.

The actuation of the whole syndrome has to be projected to the hypothalamus, to the region of the vasomotor heat-regulating centers. Here, in the ventral gray matter of the third ventricle, is situated the coöperative unit: the center of Karplus-Kreidl and the heat-regulating mechanism. Starting from this point, the pathological vasoconstrictor impulses are sent to the vessels of the brain and the organs of sense.

Thus, after all, the primary principle of the syndrome is the dysfunction of the autonomic central nervous organs.

Sclerosis pigmentosa chorioretinalis therefore is not an independent syndrome but only a *complex of symptoms*. It is the manifestation of a malignant sclerotic vascular process restricted to the brain and the sense organs. It is actuated centrally in the hypothalamus, in the ventral gray matter of the third ventricle. The affection itself is hereditary and is already manifest in the germinal anlage.

I wish to express my gratitude to Dr. med. Kurt Löwenstein, former chief of the Neurological Department in the Hospital Berlin-Lankwitz, now at Tel-Aviv, for his great interest and stimulating criticism which so greatly furthered the crystallization of my ideas.

40 Balfour Street.

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FAMILIAL PROGRESSIVE JUVENILE CATARACTS (PARATHYROID DEFICIENCY)*

ROBERT VON DER HEYDT, M.D.
Chicago

During the past five years three children of a family group of eight suffered rapidly progressive lens changes which seriously interfered with their school work.

The first, Leonard L., aged 11 years, was sent to me in May, 1934. His right eye showed an acute cortical intumescence with vision reduced to perceiving motion. The vision in the left eye was 20/100. The fundus and vitreous were normal. There was no history of skin lesions, spasms, tetany, or irradiation therapy, nor were there any symptoms of myotonic dystrophy. The following tests gave negative results: urine, bloodsugar, and Wassermann. Discissions in both lenses were uneventful and resulted in normal vision. The slitlamp showed the usual condition following discission, and no other pathologic effects.

Eleanor L., aged 21 years, an older sister, came under observation in May, 1935. The vision of the right eye under a cycloplegic was 20/60 with +4.00 D. sph.; that of the left eye was 20/150 with +3.00 D.sph. Two months previous to examination she had had a thyroidectomy. The history indicates that the lens changes antedated this operation. Her fundi were normal. Blood Wassermann and Mantoux tests were negative. Discissions resulted in normal visual acuity in both eyes, and uneventful convalescence.

In March, 1938, a younger brother, Joseph L., aged 12 years, complained of increasing difficulty with his school work. His vision was: R.E. = 20/100 and L.E. = 20/60. The fundi were normal, and, except for the lens changes, the slitlamp showed a normal cornea, iris, and vitreous.

Because of rapid progression in the development of the cataract, a discission was performed on the right eye in the fall of 1938. Normal vision resulted. The vision of the left eye deteriorated to 20/100.

The father of these children is of Polish nationality, and the mother is of German descent. The grandparents, of whom three are living, the parents, and their eight brothers and six sisters all have normal eyes.

The eight children in this family seem robust and healthy.

A careful survey of the ophthalmic literature failed to disclose any records of juveniles in whom familial cataracts were rapidly progressive.

Dr. S. Gifford reported a group of four siblings which he had seen with his father, who were similarly affected with this form of cataract. They were successfully operated on when they were 14, 24, 22, and 38 years old, respectively.

Slitlamp findings of lenses. A thin layer of dustlike spots, crystals, and some flat vacuolar changes were seen directly under the anterior and posterior capsules. There was an increased visibility of the normal subcapsular lens fiber and suture pattern. These findings are characteristic of tetany cataracts. The changes were more marked in the posterior axial cortex, but, according to Knuesel, this feature is not characteristic of tetany cataracts alone, but is seen in all complicated cataracts.

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To this precise clinical picture of tetany cataract Vogt has added the statement that these lenses seem flat and present an early sclerosis. The anteroposterior flattening of the lens was noted definitely in the cases of the two boys here described. This observation was made on the second boy four years after a similar finding was detected in the first patient.

The flattening of the lens—possibly an inhibition, or at least a retardation, of the normal lens-fiber development, due to parathyroid insufficiency—may be expected to produce a hypermetropia. If this occurred in these lenses, its effects could have been neutralized by the sclerosis, as recorded by Vogt, for in all three patients the postoperative lens replacement called for the usual difference of 10 D.

Similar disturbances of lens-fiber metabolism give rise to the related zonular, lamellar, or perinuclear cataracts. In this type of cataract the fibers involved at the period of insult to the epithelial lens metabolism are subcapsular, hence there is an apposition of clear fibers after the insult has abated. I have often seen lenses in which the metabolic disurbance was sufficiently severe to cause adhesion of the pathologically changed lens fibers to the capsule, precluding the interposition of new clear fibers. This manifestation was usually observed as a posterior change. Therefore, when examined with the narrow beam of the slitlamp, we see a quite flat, more or less opaque lens.

A subcapsular lens change of the tetany type may initiate the stormy onset of dinitrophenol cataract. Dinitrophenol is believed to exert a toxic effect on the parathyroids.

Fig. 1 (von der Heydt). The family group showing the healthy members as well as the three with progressive cataracts. The latter after successful operations.

I diagnosed as tetany cataract the incipient stage of my first dinitrophenol cataract, and had calcium and phosphorus tests made in 1935. This was before the clinical entity of dinitrophenol cataract had been recognized. Dr. Spaeth records the case of a patient in whom tetany developed long after the dinitrophenol cataracts had been removed.

In my patients all lenses, in addition to the changes observed in tetany, showed the toxic manifestations of complicated cataract at the posterior pole. As is usual in these cases, the involvement and pro-

gression do not follow anatomic lines in the lens structure. This peculiarity can be explained only on the basis of an absorption, through the thin posterior capsule, of material toxic to the lens.

Eyes afflicted with choroiditis, progressive myopia, retinitis pigmentosa, retinal separation, or intraocular tumors, as well as senile eyes, present these changes. What the factor is, presumably a toxic one, that effects so wide a variety of conditions and is common to all of them, has not yet been established.

Recent tests for blood-serum calcium in my three patients showed a content of 9.25, 8.9, and 7.6 mg., respectively. The last low figure was rechecked and established as correct. The phosphorus content was 4.47, 4.53, and 4.7. The relatively high phosphorus content is believed to have been the balancing factor that has kept these individuals free from tetany.

The children here under discussion no doubt present a recessive hereditary parathyroid-deficiency factor of sufficiently severe degree to give rise to cataracts of the tetany type.

The rarity of familial rapidly progressive cataracts in juveniles prompted the presentation of this report.

25 East Washington Street.

A STUDY OF THE INTERRELATIONSHIP OF RETINAL AND SYSTEMIC ARTERIAL PRESSURES AND INTRAOCULAR TENSION IN NORMAL AND SYPHILITIC PATIENTS*

ELIZABETH F. CONSTANTINE, M.D.
Baltimore

Since 1934, Lauber and Sobanski in Warsaw have published a total of 21 papers[1–21] dealing with the interrelationship of systemic and retinal blood pressures and intraocular tension in a variety of ophthalmological conditions such as glaucoma, choked disc, and optic atrophy. They assumed that these conditions result from disturbed nutrition of the optic nerve and retina due to disturbances in that interrelationship, and proceeded to study the retinal blood pressure in a number of normal and diseased eyes, using the ophthalmodynamometric method of Bailliart. This method will be described in detail later.

In normal eyes, Sobanski found that the retinal systolic arterial pressure ranged from 68 to 90 mm. Hg, and the diastolic arterial pressure from 40 to 50 mm. Hg. Therefore, he said, the lowest level of diastolic arterial pressure lies at least 14 mm. Hg above the highest normal level of intraocular tension (26 mm. Hg). This differential, according to Lauber and Sobanski, is the necessary condition for normal circulation in the retina. If it is diminished, there results a disturbance of the capillary circulation in the retina. The level of capillary circulation cannot be accurately determined, but is assumed to be above the level of intraocular tension and below that of retinal diastolic arterial pressure and therefore, according to Sobanski, between a mean of 33 and 48 mm. Hg.

Twelve of the above-mentioned 21 papers are devoted, at least in part, to a consideration of tabetic optic atrophy. In

* From the Wilmer Ophthalmological Institute of the Johns Hopkins Hospital and University.

this condition, Lauber and Sobanski base their argument on the assumption that all tabetics with primary optic atrophy suffer from peripheral hypotension (diastolic if not systolic). This is accompanied by a lowering in the retinal diastolic arterial pressure (normally 40 to 56 mm. Hg), which then approaches the level of intraocular tension (18 to 26 mm. Hg). When the differential between these two levels falls much below 14 mm. Hg, circulatory failure in the optic nerve occurs, producing optic atrophy. Patients with tabes who do not have optic atrophy, they argue, do not have peripheral hypotension. Therefore, logically, all tabetics with normal or elevated peripheral blood pressure should be spared from the development of optic atrophy, while all those with lowered peripheral blood pressure should proceed to develop it. They also state that all drugs used in the routine treatment of syphilis—arsenic, bismuth, mercury, and the iodides—lower systemic blood pressure, as does fever therapy, and are therefore contraindicated in cases with optic atrophy. They go on to advocate treatment based on the above theory; namely, miotics and, if necessary, operation to reduce the level of intraocular tension, thereby increasing the differential between it and the retinal diastolic arterial pressure.

In a group of 33 tabetic patients with optic atrophy, Sobanski found that this differential was 18 mm. Hg or less in 29, but below 14 mm. Hg in only 9; while in 9 tabetics without optic atrophy, it was 19 mm. Hg or more in all.

Since Lauber and Sobanski began publishing their work, a number of other workers have attempted to confirm their results. Among those who accepted their theory and corroborated their findings are Rintelen,[22] Arruga,[23] and Miklos.[24] Others have failed to confirm their figures, and state that there is no basis for their claim that tabetics suffer from hypotension and a lowered differential between retinal diastolic pressure and intraocular tension. In the latter group are Albrich and Kukan,[25] Baratta,[26] Aumann, Brüning, and Sohr,[27] and Ascher.[28] Also, there seems to be no basis for the claim of Lauber and Sobanski that the drugs used in routine antisyphilitic treatment lower blood pressure; Moore[29] states that they have no more than a transitory effect (a few minutes following injection) on the peripheral tension, in either direction. Langhammerová[30] studied the incidence of peripheral hypotension in tabetics, and found that it was four times greater in those without optic atrophy than in those with optic atrophy. Knobloch[31] attacked the problem from the experimental standpoint and after producing hypotension in rabbits, tried to cause optic atrophy by poisoning with quinine and by repeated bleeding, without results.

In an effort to study this question the following investigation was undertaken on patients under treatment in the dispensary of the Johns Hopkins Hospital. The intraocular tension, retinal arterial systolic and diastolic pressures, and systemic systolic and diastolic pressures were measured in four groups of patients, and a study was made of their interrelationship. The groups of patients studied were as follows: (1) patients between the ages of 15 and 40 years, with normal eyes; (2) patients with neurosyphilis but without optic atrophy; (3) patients with neurosyphilis and primary optic atrophy; (4) patients with neurosyphilis who had shown an ocular reaction to tryparsamide therapy. In the first group, for the purpose of examination, the pupils were dilated with homatropine, and in the last three groups with homatropine or euphthalmine. Patients with neurosyphilis were subdivided into three classes in each group: (1) tabetics and taboparetics; (2)

CHART 1—NORMALS

Patient	Age	Intraocular Tension (mm. Hg, Schiötz)	Brachial Blood Pressure (mm. Hg)		Ophthalmodynamometer Readings (gm.)		Estimated Retinal Blood Pressure (mm. Hg)		Difference between Retinal Diastolic Pressure and Intraocular Tension
			Systolic	Diastolic	Systolic	Diastolic	Systolic	Diastolic	
1	29	R 16 L 18	116	72	R 100 L 95	40 38	R 92 L 93	43 44	27 26
2	27	R 10 L 9	112	70	R 105 L 115	48 58			
3	37	R 14 L 15	120	72	R 110 L 108	52 54	R 91 L 93	51 55	37 40
4	20	R 15 L 14	104	70	R 140 L 130	62 58	R L	62 57	47 43
5	18	R 17 L 17	106	68	R 88 L 105	38 35	R 85 L 96	42 38	25 21
6	27	R 18 L 18	110	75	R 95 L 98	38 38	R 93 L 96	44 44	26 26
7	31	R 18 L 18	114	70	R 128 L 132	48 50	R L	53 56	35 38
8	15	R 17 L 19	118	70	R 110 L 110	45 45	R 99 L 103	49 52	32 33
9	32	R 11 L 13	110	68	R 125 L 125	42 42			26
10	15	R 14 L 14	108	64	R 95 L 100	35 40	R 83 L 87	33 39	19 25
11	17	R 17 L 17	112	68	R 110 L 115	44 50	R 99 L 102	48 54	31 37
12	20	R 12 L 12	110	74	R 120 L 120	48 48	R L	44 44	32 32
13	34	R 14 L 14	132	76	R 135 L 140	58 58	R L	57 57	43 43
14	19	R 19 L 19	114	66	R 92 L 90	40 38	R 92 L 91	48 46	29 27
15	24	R 19 L 19	108	68	R 95 L 95	40 40	R 95 L 95	48 48	29 29
16	37	R 15 L 15	116	76	R 125 L 120	42 40	R L	42 41	27 26
17	30	R 13 L 13	114	72	R 115 L 105	40 35	R 92 L 88	38 32	25 19
18	24	R 13 L 15	122	70	R 120 L 130	40 45	R L	38 46	25 31
19	19	R 14 L 12	110	72	R 95 L 92	38 42	R 83 L 77	37 38	23 26
20	38	R 22 L 22	104	72	R 105 L 110	40 40	R 107 L 108	52 52	30 30

CHART 1—*Continued.*

Patient	Age	Intraocular Tension (mm. Hg Schiötz)	Brachial Blood Pressure (mm. Hg)		Ophthalmodynamometer Readings (gm.)		Estimated Retinal Blood Pressure (mm. Hg)		Difference between Retinal Diastolic Pressure and Intraocular Tension
			Systolic	Diastolic	Systolic	Diastolic	Systolic	Diastolic	
21	40	R 15 L 16	130	88	R 140 L 130	65 62	R L	65 64	50 48
22	34	R 13 L 14	106	66	R 100 L 95	30 30	R 85 L 83	28 29	15 15
23	38	R 16 L 15	108	70	R 95 L 90	36 38	R 90 L 82	38 39	22 24
24	38	R 21 L 21	122	82	R 100 L 105	45 42	R 104 L 107	56 53	35 32
25	37	R 16 L 16	128	75	R 105 L 110	45 48	R 94 L 97	48 51	32 35
26	37	R 22 L 22	120	82	R 120 L 115	60 60	R L 112	72 72	50 50
27	26	R 17 L 15	125	84	R 100 L 98	40 45	R 94 L 88	44 46	27 31
28	38	R 17 L 17	130	84	R 130 L 125	42 50	R L	46 54	29 37
29	25	R 12 L 14	122	68	R 98 L 98	38 38	R 80 L 85	33 37	21 23
30	23	R 14 L 17	112	66	R 100 L 95	45 45	R 87 L 90	44 49	30 32
31	26	R 19 L 17	130	82	R 80 L 75	40 35	R 83 L 77	48 38	29 21
32	35	R 18 L 25	114	66	R 118 L 120	50 42	R L	56 57	38 32
33	23	R 13 L 14	110	74	R 78 L 78	42 36	R 73 L 75	39 35	26 21
34	38	R 18 L 18	125	86	R 82 L 88	38 40	R 83 L 88	44 46	26 28
35	29	R 17 L 17	130	80	R 85 L 82	35 38	R 83 L 82	38 42	21 25
36	27	R 12 L 15	104	68	R 70 L 75	25 30	R 65 L 73	22 30	10 15
37	40	R 12 L 15	102	66	R 100 L 95	45 42	R 82 L 85	41 42	29 27
38	38	R 15 L 14	115	76	R 100 L 100	38 38	R 89 L 87	39 37	24 23
Range	15–40	9–25	102–132	64–88	70–140	25–65	65–112	28–72	10–50
Average	29	16	116	73	106	43	89	46	29

paretics; (3) those with diffuse meningovascular syphilis. All the syphilitic patients had received or were receiving routine antisyphilitic therapy, and some had been given induced-malaria or Swift-Ellis treatments. Patients were placed in a semirecumbent position and remained

CHART 2

CENTRAL NERVOUS SYSTEM SYPHILIS—NO OPTIC ATROPHY

Patient	Type of Syphilis	Age	Intraocular Tension (mm. Hg Schiötz)	Brachial Blood Pressure (mm. Hg)		Ophthalmodynamometer Readings (gm.)		Estimated Retinal Blood Pressure (mm. Hg)		Differential between Retinal Diastolic Pressure and Intraocular Tension
				Systolic	Diastolic	Systolic	Diastolic	Systolic	Diastolic	
1	Taboparesis	37	R 13 L 11	114	84	R 98 L 105	35 45	R 83 L	32	19
2	"	59	R 14 L 14	146	92	R 135 L 130	65 60	R L	63 59	49 45
3	Paresis	37	R 12 L 12	100	72	R 110 L 105	50 48	R 87 L 85	46 44	34 32
4	Diffuse meningovascular syphilis	51	R 18 L 17	116	76	R 125 L 120	55 50	R L	61 54	43 37
5	"	38	R 17 L 15	116	86	R 85 L 80	30 22	R 83 L 78	32 23	15 8
6	"	54	R 15 L 14	110	75	R 128 L 130	58 55	R L	58 53	43 39
7	"	28	R 17 L 17	116	72	R 118 L 128	48 55	R L	52 59	35 42
8	"	43	R 19 L 22	122	92	R 120 L 118	48 45	R L	55 57	36 35
Range		28–59	11–22	100–146	72–92	80–135	22–65	78–87	23–63	8–49
Average		43	15	118	81	115	48	83	50	34

so for all measurements. First the intraocular tension was measured with the Schiötz tonometer. The systemic blood pressure was taken in all cases on the right arm. The retinal pressures were then measured by the method described by Bailliart in his monograph on the subject.[32] Bailliart's ophthalmodynamometer was placed on the sclera inside the outer canthus, and pressure applied to the globe as nearly as possible in a horizontal plane and perpendicularly to the globe. By direct ophthalmoscopic examination the arteries were observed as they emerged from the disc, and the pressure at which pulsation first appeared was taken as the diastolic pressure. According to Bailliart, "theoretically it is the maximum pulsation that should denote the diastolic pressure. Practically there is so little difference between this and the first visible arterial pulse that, for the retinal artery, the latter can be considered a proper criterion."[33] The pressure at which the arterial pulse is obliterated was taken as the

CHART 3

CENTRAL NERVOUS SYSTEM SYPHILIS—WITH PRIMARY OPTIC ATROPHY

Patient	Type of Syphilis	Age	Intraocular Tension (mm. Hg Schiötz)	Brachial Blood Pressure (mm. Hg)		Ophthalmodynamometer Readings (gm.)		Estimated Retinal Blood Pressure (mm. Hg)		Differential between Retinal Diastolic Pressure and Intraocular Tension
				Systolic	Diastolic	Systolic	Diastolic	Systolic	Diastolic	
1	Tabes	34	R 18 L 17	104	70	R 88 L 88	32 34	R 88 L 85	37 37	19 20
2	"	38	R 17 L 17	120	82	R 120 L 120	60 65	R L	64 68	47 51
3	"	43	R 22 L 22	148	85	R 125 L 115	55 40	R L 111	67 52	45 30
4	"	49	R 25 L 25	116	80	R 100 L 100	45 40	R 109 L 109	60 55	35 30
5	"	57	R 17 L 17	116	76	R 115 L 120	48 52	R 102 L	52 56	35 39
6	"	70	R 22 L 17	148	80	R 105 L 110	48 50	R 107 L 99	59 54	37 37
7	Taboparesis	63	R 14 L 16	170	95	R 120 L 110	40 36	R L 97	39 38	25 22
8	"	46	R 18 L 15	120	72	R 88 L 100	35 38	R 88 L 89	40 39	22 24
9	"	35	R 16 L 16	105	60	R 95 L 95	40 38	R 90 L 90	43 40	27 24
10	"	49	R 11 L 14	135	90	R 135 L 140	60 65	L	63	49
11	Diffuse meningovascular syphilis	61	R 19 L 19	142	74	R 120 L 120	40 40	R L	48 48	28 28
12	"	69	R 20 L 20	155	105	R 140 L 145	78 85	R L	83 89	63 69
13	"	44	R 11 L 11	108	72	R 95 L 95	40 38			
14	"	31	R 13 L 15	108	80	R 115 L 110	55 50	R 92 L 94	51 51	38 36
15	"	45	R 11 L 13	108	68	R 85 L 85	28 30	L 76	28	15
16	"	59	R 16 L 14	185	120	R 150 L 150	78 85	R L	78 78	62 64
Range		31–70	11–25	104–185	60–120	85–150	28–85	76–111	28–89	15–69
Average		50	17	130	82	112	49	95	54	36

systolic pressure. The ophthalmodynamometer is calibrated in grams water, and the readings obtained indicated only the pressure applied to the eyeball, and not the pressure transmitted to the retina. The determination of the latter presents difficulties, as various factors enter in: not only is the initial intraocular tension to be considered, but also the elasticity of the ocular tunics, the actual area over which the pressure is applied to the eyeball, and other factors. No entirely satisfactory method of correlating these factors appears in the literature, and for the purposes of this investigation Bailliart's method has been used. As it was also used by Lauber and Sobanski, results should be comparable with theirs. Bailliart performed a series of experiments on cats, in which a needle connected with a manometer was introduced into the anterior chamber, and readings taken as the pressure with the dynamometer was varied. From these results he constructed a graph,[34] on which the pressure transmitted to the retina is read directly in mm. Hg, given the initial intraocular tension in mm. Hg (Schiötz), and the ophthalmodynamometer reading in grams water. However, his graph does not cover the entire range of readings found in the patients studied, and in cases with an initial intraocular tension below 12 mm. Hg, or an ophthalmodynamometer reading over 115 gm. water, no attempt was made to estimate the transmitted pressure.

CHART 4

CENTRAL NERVOUS SYSTEM SYPHILIS—OPHTHALMIC REACTION TO TRYPARSAMIDE THERAPY

Patient	Type of Syphilis	Age	Intraocular Tension (mm. Hg Schiötz)	Brachial Blood Pressure (mm. Hg)		Ophthalmodynamometer Readings (gm.)		Estimated Retinal Blood Pressure (mm. Hg)		Differential between Retinal Diastolic Pressure and Intraocular Tension
				Systolic	Diastolic	Systolic	Diastolic	Systolic	Diastolic	
1	Paresis	68	R 13 L 13	105	75	R 105 L 105	58 50	R 88 L 88	54 47	41 34
2	"	59	R 14 L 16	142	70	R 120 L 120	40 38	R L	39 40	25 24
3	Diffuse meningovascular syphilis	70	R 16 L 15	195	68	R 115 L 115	30 32	R 99 L 97	31 32	15 17
4	"	66	R 13 L 15	115	76	R 120 L 115	48 45	R L 97	46 46	33 31
5	"	48	R 21 L 21	150	95	R 135 L 135	55 55	R L	66 66	45 45
Range		48–70	13–21	105–195	70–95	105–135	30–58	88–99	31–66	15–45
Average		62	16	141	77	118	45	94	47	31

Bailliart found that in normal eyes the average retinal blood pressure was: systolic, 65 to 70 mm. Hg, diastolic, 30 to 35 mm. Hg. He gives the ratio of retinal to systemic systolic pressure as .54, and of retinal to systemic diastolic pressure as .45.

The results obtained in the four groups

CHART 5

SUMMARY

Class of Patient	No.	Average Age	Average Intra-Ocular Tension	Average Brachial Blood Pressure		Average Estimated Retinal Blood Pressure		Average Differential between Retinal Diastolic Pressure and Intraocular Tension
				Systolic	Diastolic	Systolic	Diastolic	
Normals	38	29	16	116	73	89	46	29
Neurosyphilis, no optic atrophy	8	43	15	118	81	83	50	34
Neurosyphilis, with primary optic atrophy	16	50	17	130	82	95	54	36
Neurosyphilis, with tryparsamide reaction	5	62	16	141	77	94	47	31

of patients are here tabulated in charts 1, 2, 3, and 4. These charts give the fundamental data on each of the various patients. Chart 5 gives a comparison of the mean readings of the four different classes of patients.

COMMENT

The readings with the ophthalmodynamometer here reported are uniformly higher than those obtained by Bailliart. They are, however, approximately the same as those reported by Lauber and Sobanski. As has been pointed out by other observers, individual variations in the manner in which the ophthalmodynamometer is applied are responsible for such differences. The method is palpably quite inaccurate and open to wide differences, depending on the personal equation of the observer. Since the figures obtained in these patients are consistent throughout for the various classes of patients, any error due to the personal equation would be constant throughout and not influence the conclusions.

A study of chart 5 shows, first, that the patients in this series with central-nervous-system syphilis did not have a peripheral vascular hypotension. On the contrary, the average systemic blood pressure of the 29 syphilitic patients in groups II, III, and IV, was 129/80 mm. of mercury, while in 38 normal individuals the average pressure was 116/73 mm. The difference in the mean ages of these two groups, 52 years and 29 years, respectively, is of course quite sufficient to account for this slightly higher blood pressure in the syphilitic group. Moreover, among the syphilitic patients, 8 individuals with no optic atrophy and a mean age of 43 years showed an average systemic blood pressure of 118/81 mm., while 16 syphilitic patients with primary optic atrophy, and a mean age of 50 years, had an average blood pressure of 130/82. Quite obviously from these figures there is no substantiation of Lauber's and Sobanski's claims that individuals with central-nervous-system syphilis and optic atrophy have a relative vascular hypotension.

The readings with the ophthalmodynamometer and estimated retinal systolic and diastolic blood pressures are in

accord with the absence of vascular hypotension in the syphilitic patients. The average retinal pressure was definitely higher in the syphilitic patients than in the normal individuals. In fact, the 16 patients with central-nervous-system syphilis and optic atrophy had the highest systolic and diastolic pressure of any of the four groups.

The final and fundamental point in the theory of Lauber and Sobanski is that the primary optic atrophy is dependent upon a low difference between the retinal diastolic pressure and the intraocular tension. The evidence in this study is directly to the contrary. The mean differential in the normal individuals was 29 mm., in syphilitics without optic atrophy it was 34 mm., and in syphilitics with optic atrophy, 36 mm., the highest figure of all. Furthermore, the patients with central-nervous-system syphilis who had a tryparsamide reaction with involvement of the optic nerves showed a normal differential between the retinal diastolic pressure and the intraocular pressure. Finally, a study of the individual cases in charts 1, 2, and 3 gives no support to Lauber's and Sobanski's theory. In but one patient with syphilis and optic atrophy (group III), was the differential below 19, and in this instance it was 15. In groups I and II, normal individuals and syphilitics without optic atrophy, there were 6 cases in which the differential was below 19, in one instance as low as 8, yet with no evidence of atrophy of the optic nerve.

Summary

A study of the intraocular tension, the peripheral systemic systolic and diastolic blood pressures, and the retinal systolic and diastolic blood pressures in (a) 38 normal individuals, (b) 8 patients with central-nervous-system syphilis, but without optic atrophy, (c) 16 patients with central-nervous-system syphilis with primary optic atrophy; and (d) 5 patients with central-nervous-system syphilis who had an ophthalmic reaction from tryparsamide therapy, gives no support to the theory of Lauber and Sobanski on the pathogenesis of optic atrophy. On the contrary, the results are in direct contradiction to their theory. No peripheral vascular or retinal hypotension was observed in patients with central-nervous-system syphilis, either with or without optic atrophy. The differential between the retinal diastolic pressure and the intraocular pressure was not reduced in patients with optic atrophy. On the contrary, it was higher in these patients than in normal controls.

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NOTES, CASES, INSTRUMENTS

OCULAR SYMPTOMS IN NASOPHARYNGEAL TUMORS

GUERDAN HARDY, M.D.
Saint Louis

Fortunately, tumors of the nasopharynx are not frequent but those that do occur are usually not diagnosed until late and then generally after cervical metastases have taken place. Obviously more hope can be offered to those in whom the condition is recognized early. I wish to stress the importance of a thorough examination of the nasopharynx in those cases of ocular palsy and proptosis in which no obvious cause is manifest.

Practically, one may divide the symptoms and signs into two groups, the nasopharyngeal and the extranasopharyngeal. The nasopharyngeal symptoms are often late, with the possible exception of nosebleed. Nasal obstruction when it occurs is almost always in the advanced case.

The extranasopharyngeal symptoms may be aural, such as fullness in the ear, tinnitis, and deafness. Frequently, however, the symptoms and signs are orbital. Enlarged neck glands appear soon, for metastases occur early.

Since the site of origin of such tumors is near the sphenoidal fissure and the apex of the orbit, we find the following manifestations: The sixth nerve may be involved as it passes through the sphenoidal fissure. Extension of the malignancy along this same route may give rise to pain in and around the eye, diplopia, proptosis, ptosis, papilledema, and later optic atrophy. Other cranial nerves may also be involved, all of which adds confusion to the clinical picture. Thus the second, third, fourth, and the maxillary division of the fifth may be paralyzed. Posterior extension does not take place frequently but when it does symptoms and signs of brain tumor develop.

Hansel[1] found symptoms referable to the eye in eight of the nine cases he reported. Woltman[2] described sixth-nerve paralysis in 18 of 25 cases. According to Hansel the cranial nerves are usually involved extracranially. He found the second nerve involved in three cases, the third in four, the fourth in three, and the sixth in five instances.

The growths in the nasopharynx are frequently undifferentiated, rapidly growing, and show in section many mitotic figures. They may be squamous-cell epithelioma, carcinoma, and various forms of sarcoma.

CASE REPORT

A colored man, 57 years old, came to the Saint Louis County Hospital Clinic on January 6, 1938. He complained of pain in the left side of the face, and of left temporal headache. His symptoms had developed two weeks before this and presumably followed an upper respiratory infection. Examination revealed a vascular hypertension, arteriosclerosis, and a paralysis of the left external rectus. Because of the latter finding he was referred to the eye clinic, where he was examined by Dr. Dan Bisno on January 24, 1938.

Dr. Bisno confirmed this observation. Nothing else unusual was noted about the eyes except a sluggishness of the pupil of the left eye. A blood Wassermann was requested, and he was referred to the neurological clinic.

Neurological examination on January 27th was essentially negative except for a suggestive lower left facial palsy. A tentative diagnosis of cerebral arterio-

sclerosis with a small pontine thrombosis was made. The blood Wassermann test proved to be negative.

On February 10, 1938, a paralysis of the left third, sixth, and seventh cranial nerves, and a beginning proptosis were noted. Hospitalization for study was recommended.

The patient entered the Saint Louis County Hospital on February 12, 1938. An additional history of occasional epistaxis during the previous summer was obtained at that time. On February 17th he was examined by Dr. Carl Beisbarth, who reported the fundus of the left eye normal and its vision good. By this time there was complete left ophthalmoplegia.

Because of hazy sinuses in a routine X-ray film made shortly after admission, I was asked to examine the patient. A fairly large tumor in the left part of the nasopharynx was found. Biopsy sections showed the growth to be a squamous-cell carcinoma with a few mitotic figures. Since the tumor was in an inoperable location radiation was recommended but, for reasons not known to me, it was not given. During the next few weeks he became steadily worse and expired March 18, 1938.

Summary

The early appearance of ocular and orbital signs in the presence of tumors of the nasopharynx has been emphasized. Early recognition is important since these growths are sometimes extremely radiosensitive. Once extension or metastases have occurred the condition is hopeless.

Humboldt Building.

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SIMULTANEOUS COLOR CONTRAST*

An instrument for demonstrating it

Carl T. Eber, M.D.
Saint Louis

In a comprehensive review of the literature on phenomena of color sensations presented by the author to the Committee on Theses of the American Ophthalmological Society in 1935,[1] the more important theories and contributions were discussed. In his concluding remarks he stated "that at the present time no satisfactory and complete theory of color vision can be constructed from the material contained in the voluminous literature on the subject, including the pertinent material furnished by allied branches of science. . . . Researches on photoelectric response in the rods and cones, the existence of reflex processes, experimental findings in the phenomena of induction, and simultaneous color contrast have proved definitely to be an advancement toward a desirable and consistent solution. The clinical application of some of these findings to abnormal persons may help to prove or disprove their value."

All visual experiences, except the depth factor, may be classed under visual color sensations—chromatic and achromatic. Mills[2] thinks that "the centers for color

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*Read before the Saint Louis Ophthalmic Society, March 25, 1938. A report of the proceedings of this meeting appeared in the April, 1939 issue of this Journal with an interesting discussion of this paper by Father H. Gruender, S.J.

(recognition) are contiguous to the primitive (luminosity) centers for sight." We acknowledge certain laws in the behavior of physical light and colors, but the psychologic evaluation is quite different. We are ignorant of the mechanism which transforms physical light into nerve currents and the manner in which these so-called "nerve-currents" are transformed into consciousness. Where are the centers for colors? How are they arranged? How do we recognize innumerable hues? Why do some individuals recognize many more hues than others? These and many more pertinent questions must remain unanswered in our present state of knowledge. Numerous theories and alterations in existing theories have been advanced, but they have all failed because of "incomplete consideration of the entire visual process."

Though simultaneous contrast has been recognized for many years and crudely demonstrated, only in recent years has the psychologic aspect of color vision received more serious attention. By simultaneous contrast we refer to a visual sensation (chromatic or achromatic) in which a part of the field is influenced or inhibited simultaneously by another contiguous field on the retina. "Afterimages" are produced by successive visual stimuli. The psychologic response to physical stimulation, of necessity, is observational and cannot be explained on a physical basis. Examples of this are: (1) Brightness contrast—the change of an achromatic sensation to one of another shade by merely changing the surrounding (inhibiting) achromatic light intensity; (2) saturation contrast—the changing of a chromatic shade even to an achromatic (black) by a surrounding area of the same kind of chromatic light but of greater intensity, due to partial inhibition; (3) the two forms of color contrast—in the first form, the change of a neutral color into a chromatic one complementary to the inhibiting chromatic light; in the second form, partial inhibition of chromatic light by a noncomplementary chromatic light producing a new color sensation.

As stated by Gruender,[3] inhibition is responsible for all of these forms of contrast; that is, color sensations from specifically stimulated areas on the retina are altered by contiguous retinal areas that are stimulated by different light values. This effect is caused by nervous inhibition. He demonstrates these phenomena by means of a contrast box the construction of which was suggested by Hering's *Doppelschirmanordnung* briefly described in Froebe's "Lehrbuch der experimentellen Psychologie," vol. I, p. 67. Gruender's contrast box is described in his book on "Experimental psychology." The author's instrument[4] is a modification of Gruender's and was presented and described before the American Ophthalmological Society in June, 1937. The essential changes are: (1) Artificial light source, well approaching that of daylight in place of variable natural light; (2) two sets of chambers instead of one, making the instrument available for binocular observations; (3) variable shutters and diaphragms to control the intensity of light, and appropriate scales to measure the amount of light admitted to the chambers in foot-candles; (4) specially selected glass light filters of known spectral values instead of gelatin filters of unknown values.

Up to the present time, only normal eyes have been employed in experiments to demonstrate the various forms of color contrast. The results with this instrument are essentially the same as those recorded by Gruender,[5] "but the relative quantities of white and filtered light to produce specific contrast effects are different because of the difference in the filters, dia-

phragms, sources of light, light measurements, and construction between the two instruments."

In a small series of observations, the following averages were obtained (see fig. 1).

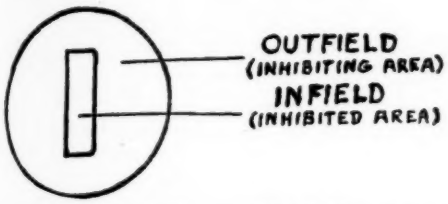

Fig. 1 (Eber). Diagram of infield and outfield as seen in the author's instrument. Each of the two fields in the instrument is illuminated independently and arranged as above.

Brightness contrast: 26 ft-c of inhibiting white light in the outfield to cause 4 ft-c of white light in the infield to appear black.

Saturation contrast: 27 ft-c of inhibiting blue light in the outfield to cause 4 ft-c of blue light in the infield to appear black.

First form of color contrast: 27 ft-c of inhibiting red light in the outfield to cause 4 ft-c of white light in the infield to appear blue-green.

Second form of color contrast: 24 ft-c of inhibiting green light in the outfield to cause 4 ft-c of yellow light in the infield to appear orange-red.

Carleton Building.

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IMPLANTATION EPITHELIAL CYST IN ANTERIOR CHAMBER

Successful Removal

Martha Brewer Lyon, M.S., M.D., and William E. Miller, B.S., M.D.
South Bend, Indiana

In the accessible literature most of the cases of cyst in or on the iris or in the anterior chamber are due to implanted epithelium following injuries or operations, especially those for cataract. Reports of pearl cysts following injury with the implantation of a cilium into the anterior chamber are apparently rare; hence this case with its successful outcome seems worth reporting.

Summary of the Literature

Fuchs[1]: Pearl cysts . . . probably arise through implantation of torn-off portions of epithelium into the iris. . . . The finding of hair indicates that this epithelium arose from the skin.

Roemer[2]: Pearl cysts, which are met with more rarely, are firm tumors in either the tissue or the surface of the iris. . . . They likewise follow an injury usually when a cilium has been carried into the anterior chamber, and months and years may elapse after the injury before the tumors begin to grow. Epithelial cells coming from the epidermis, from the root sheaths and cells of the hair bulbs of the cilia, or from the epithelium of the cornea are torn away at the time of the accident and transplanted into the iris where they proliferate.

Foster[3]: [A cyst of the iris] . . . when it has the luster of mother of pearl is a pearl cyst which always is traumatic in its origin and is usually due to the

implantation of an eyelash into the iris . . . .

Hoffmann[4]: [Quotation from Ophthalmic Year Book] . . . subsequent to perforating injury by a piece of glass Hoffmann found on the iris three lashes which were removed. After eight months a pearl cyst developed connected by a strand with the iris.

Roth and Geiger[5]: . . . history of perforating injury—a piece of steel which was removed with a magnet 20 years previously . . . by means of a slitlamp . . . a cilium could be seen in the cyst wall.

Collins and Mayou[6]: Epithelial pearl tumors may form around an eyelash when it becomes implanted in the interior of the eye; they arise from the cells of its root sheath.

Krug[7]: If particles of skin or hair enter the eye, a firmer tnmor of epidermoid cells is formed, namely a pearl cyst, cholesteatomatous or granulomatous in character. Such tumors are usually found in the anterior chamber.

Most successful treatment of the so-called pearl cyst has been surgical, in which the cyst including the implanted cilium has been removed by an iridectomy.

Case History

On August 26, 1937, J. D., a boy of 12 years, while using an auger to bore holes, attempted to pull the augur out too rapidly. The point of the augur flew up cutting the cornea of the right eye at about the 4-o'clock position. As the eye was not painful, it was not given much attention until the next evening at 7:30, when a doctor was called because the boy had a fever of 104 degrees from an attack of acute tonsillitis. At this time, the doctor was asked to look at the eye, and he immediately suggested that an ophthalmologist be called. When seen later that evening (by M. B. L.), 36 hours after the accident, the iris was caught and sealed in the cut and the conjunctiva was quite red. No attempt was made to free the iris because of the tonsillitis and fever. The eye was treated expectantly with 0.5-percent atropine ointment every two hours; merthiolate ointment, 1 to 5,000, every four hours; ice compresses every hour, five minutes at a time. No infection of the eye followed the tonsillitis, but the eye was kept dilated throughout the next month. The pupil contracted normally, and vision of 20/50 was obtained. This improved under 2-percent solution of dionin to 20/32 by March. The fundus apparently was normal.

During the summer of 1938 the boy went to camp and did much high diving.

On August 17, 1938, the mother noticed a white spot in the iris for the first time, and the boy was seen the next day. There was a white, glistening, round body on the iris, 1.5 mm. in diameter, half way between the pupillary margin and the periphery of the iris not over the scar but on the opposite side of the eye in about the 8-o'clock position. Examination with the slitlamp showed a few loops of iris fibers over the cyst and a cilium extending from the temporal side of the cyst up into the anterior chamber. Under atropine the pupil dilated round with no adhesions to lens capsule and apparently no connection with the former scar. On September 19, 1938, the cyst measured 2 mm. in diameter.

Two days later, the boy was taken for consultation to Dr. Robert von der Heydt. He advised an X-ray examination for possible intraocular foreign body, also advised leaving the cyst alone and watching it. Figure 1 is a photograph of the cyst taken by Dr. von der Heydt at that time. The X-ray study later showed no foreign body.

The cyst was watched each week until

Fig. 1 (Lyon and Miller). Photograph of cyst with cilium erect and almost touching pupillary margin. Courtesy of Dr. Robert von der Heydt, September 21, 1938.

Fig. 2 (Lyon and Miller). Photograph of both eyes, July 22, 1939. Right pupil enlarged to temporal side and downward.

December 3d, when it measured 2.5 mm. in diameter, the upper border touching the margin of the pupillary area of the iris; the cilium instead of standing straight up bent in at the top toward the midline and the round pupil was distorted toward the cyst. At no time was there any apparent inflammation of nor pain in the eye.

During December the cyst grew quite rapidly, and it became a question of losing the eye because of the growth or risking its removal.

An operation was performed on Janu-

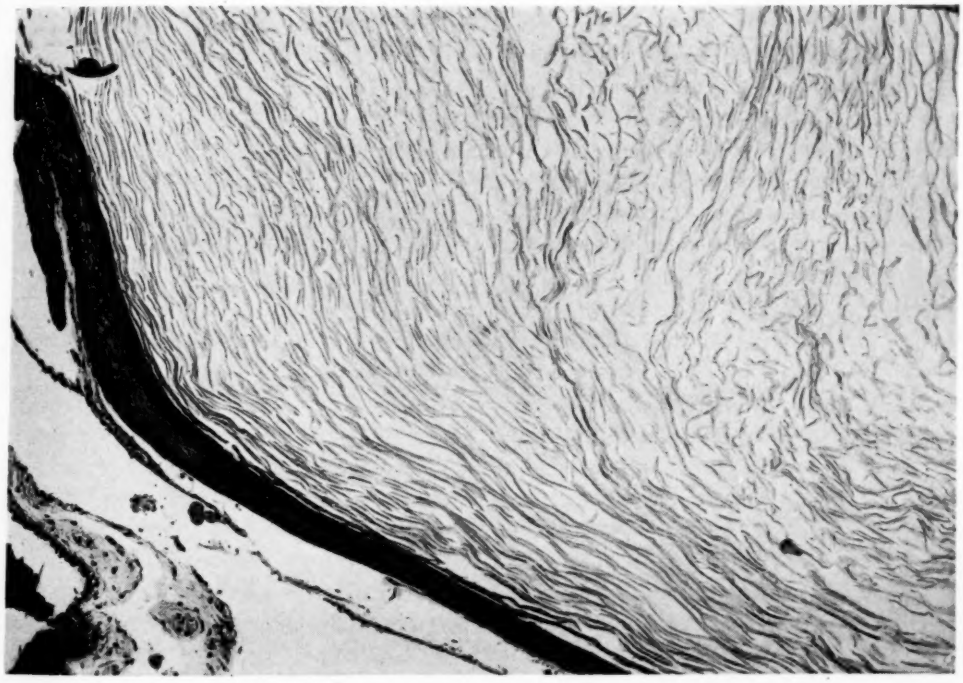

Fig. 3 (Lyon and Miller). Photomicrograph of portion of cyst, showing the layers of the skin from left upper corner to lower right border and the desquamated keratin occupying most of the picture. In the lower right hand corner are apparently tags of iris. Magnification about 100 diameters.

ary 23, 1939, under avertin and ether anesthesia (by W. E. M.).

A small keratome was used for the first incision, which was then enlarged with scissors. It was found that the cyst was quite firmly adherent to the iris and the posterior surface of the cornea. The cyst was freed below with small iris scissors, and above with a small hook, and then removed with a cataract loop. The cyst did not break and the cilium came out attached to it. A few shreds of iris were also attached. Shreds of iris appearing in the incision were cut off and the edges replaced. The incision healed without complications, and the boy (May 13th) has a good eye with a pupil elongated downward toward the temporal side at about the 7 o'clock position with vision of 20/40. Slitlamp examination showed no recurrence at this time. Figure 2 is a photograph of the eye taken on July 22, 1939.

The cyst with cilium attached was sent to the Army Medical Museum and the following report was received from Lt. Col. J. E. Ash, Medical Corps U. S. A., Curator, on February 24, 1939: The specimen is a thin encapsulated white cyst 5 x 4 x 4 mm. containing a firm, slightly translucent white material with a central opaque core about 1.0 mm. in diameter. A hair 4 mm. long was found on the external surface of the cyst wall.

The microscopic section shows a cyst lined by squamous epithelium with basal, malpighian, granular, and keratin layers of desquamated keratin and attached to iris tissue.

Diagnosis: Epithelial cyst of anterior chamber probably secondary to traumatic implantation of integumentary epithelium.

Figure 3 is a photomicrograph of a portion of the cyst showing the characteristic layers of the skin and the central mass of desquamated keratin.

806 Building and Loan Tower
201 Christman Building

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SOCIETY PROCEEDINGS

Edited by Dr. H. Rommel Hildreth

NEW YORK SOCIETY FOR CLINICAL OPHTHALMOLOGY

December 5, 1938

Dr. Percy Fridenberg, *presiding*

Neuro-ophthalmology

Dr. Daniel Kravitz gave an instructional hour on this subject.

Eye movements: voluntary-involuntary reflex, their source and significance

Prof. Alfred Bielschowsky spoke on this subject and showed how the ocular mechanism was divided into four groups: volitional, nonvolitional, reflex, and fusion. He explained the relation of the posterior longitudinal bundle to ocular movements in cases of compensatory torticollis and showed how pareses of ocular muscles were often due to supranuclear lesions rather than to those of peripheral nerves.

Discussion. Dr. Joseph Globus in commenting said that he found that the relation of the posterior longitudinal bundle to ocular movements in torticollis was a phenomenon having some of the earmarks of the Magnus and deKleyn phenomenon. Neurologists, while familiar with lesions above the bulb (pseudobulbar paralysis) affecting the tongue, mouth, and muscles of deglutition, and so forth, do not know that the eye may be similarly involved.

Dr. Morris Davidson said that there was not supposed to be any direct connection between the motor area and the extrapyramidal system. In cases of encephalitis lethargica there are always disturbances of eye motility. Hence there must be an extrapyramidal path concerned with reflex movements of the eye at least. Is it not possible that these movements which Professor Bielschowsky describes as involuntary but not reflex also pass via the extrapyramidal system?

Dr. Joseph Pascal called attention to the fixation reflex which was not mentioned by Dr. Bielschowsky; that is, when the eye fixates an object, the object in turn fixates the eye and holds it steady. He agreed with Dr. Hardy that Dr. Krimsky's definition of fusion is much too simple in view of Verhoeff's recent work, in which he showed that there is really no blending or superposition of two whole retinal images, but that the conscious image is merely a mosaic of parts of the two images.

Professor Bielschowsky in summarizing called attention to the striking resemblance between cases with disturbances of the associated eye movements and those of pseudobulbar paralysis. In Huntington's chorea, Wilson's disease, postencephalitis Parkinsonism, one comes across patients whose eyes are apparently unable to move voluntarily or at command, whereas the patients can apparently read without any difficulty. It must be borne in mind that voluntary movements are the response to impulses given from the frontal-lobe center. Involuntary movements will not occur unless an object that is seen or heard attracts attention.

He has not been able thus far to answer why head tilting gives no results in pareses of the vertical recti, while giving positive results in 100 percent of paresis or congenital deficiencies of the oblique muscles. It may be because the oblique muscles have a stronger rotating component than the vertical recti. At any rate,

a positive result with the head-tilting test where diagnosis is uncertain proves the case to be an involvement of the obliques.

Dr. Joseph Globus questioned whether encephalitis really was a disease of the extrapyramidal system and pointed out that the picture of extrapyramidal disease may result from such lesions as arteriosclerosis, degenerations, and inflammations so long as it affects the mid-brain predominantly. He also pointed out that in Parkinson's disease the oculogyric crises and so-called cogwheel movements of the eyes are not uncommon. Therefore he felt that when the extrapyramidal system is injured, as in epidemic encephalitis, the reflex eye mechanisms are often also affected.

Psychological considerations in tests of binocular function

Dr. Emanuel Krimsky in discussing this subject pointed out the great need for more intelligent orthoptic training. He felt that parents, teachers, and young patients themselves might be made to take a greater interest in this training by proper psychological approach.

Discussion. Dr. LeGrand Hardy, in commenting, observed that the whole problem of fusion and stereopsis is being oversimplified. He felt that in at least 60 percent of convergent-squint cases there is an abnormal retinal correspondence. In these cases training, unless carried out under expert supervision, is worse than useless. Only in phorias is home orthoptic training of any value.

Dr. Krimsky in summarizing agreed with Dr. Hardy that cases of abnormal retinal correspondence should not be treated in the home. But in his opinion many of the cases with normal retinal correspondence can be treated in the home adequately if followed by periodic check-ups. He believes that a few good stereoscopic pictures are better than many poor ones, often supplied with the stereoscope.

Sidney A. Fox,
Secretary.

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY

January 17, 1939

Dr. Edwin B. Goodall, *presiding*

Ocular signs and symptoms due to syphilis, especially syphilis of the nervous system

Dr. H. Houston Merritt read a very interesting paper on this subject. He said that the neurosyphilologist is prone to feel that syphilis is one of the more important causes of diseases of the eye, but patients with syphilis of the eye represent only a minute fraction of those seen by an ophthalmologist.

It was Dr. Merritt's purpose to present the known facts regarding syphilitic diseases of the eye and to point out in what respects knowledge was lacking.

Statistics as to the importance of syphilis as the cause of iritis have come chiefly from eye clinics closely associated with large syphilitic clinics and as a result figures (such as 3 percent to 40 percent) abnormally high to a practicing ophthalmologist are reported. Moore reports that involvement of the nervous system is found in only 1 percent of the patients with iritis early in the secondary stage of the disease, but that it is present in 12 percent of those with iritis in the later course of syphilis. This relatively high frequency of the coincidence of syphilis of the iris and central nervous system is important to the ophthalmologist in that he should see that an examination of the spinal fluid is made in all patients with iritis that develops in the course of syphilis.

The treatment of syphilitic iritis does

not usually present any great difficulty and consists of local treatment of the eye and routine antisyphilitic treatment (alternate courses of injections of one of the arsenicals and one of the heavier metals), continued for at least one to two years, depending upon the rapidity of the reversal of the serum Wassermann reaction and the presence or absence of abnormalities in the spinal fluid. Under this treatment, there is usually a rapid subsidence of the iritis. Fever therapy (by intravenous typhoid vaccine or by the "hot-box") has been employed with success in resistant cases. With routine treatment normal visual acuity is preserved in nearly 90 percent of the cases occurring early in the course of syphilis and in approximately 60 percent of those that develop late in the disease.

Interstitial keratitis is usually a manifestation of congenital syphilis. It occurs in the acquired form of the disease practically only as a complication of iritis. Carvill and Derby estimated that patients with interstitial keratitis represented 0.5 percent of all the admissions to an eye infirmary. On the other hand, 50 percent of the patients with congenital syphilis develop interstitial keratitis at some time. The disease may develop at any age up to 40 years, but it is most common between the first and tenth years of life. Carvill and Derby reported that routine treatment (local treatment of the eye and antisyphilitic therapy) results in preservation of normal vision in 75 percent of the cases and good vision in an additional 12 percent.

The incidence of keratitis in the other eye is diminished from 27 percent in untreated cases to 3.6 percent by treatment at the time of the involvement of the first eye (Carvill and Derby). The danger of the development of interstitial keratitis is one cogent argument for the treatment of patients with asymptomatic congenital syphilis. The occurrence of interstitial keratitis has been reported in congenital syphilitics who have previously received antisyphilitic treatment, but the rarity of such an occurrence is attested by the statistics of the clinical coöperative group.

Fever therapy is a valuable adjunct in the treatment of the resistant cases of interstitial keratitis.

Paralyses of the ocular muscles are not infrequently encountered in tabes dorsalis, and to less extent in dementia paralytica. They are a frequent manifestation of syphilis of the meninges. The symptoms of syphilis of the meninges may develop at any stage of the disease but are most common in its first year. Paralysis of the muscles supplied by the oculomotor nerve is the most frequent, with that of the abducens next, and involvement of the trochlear very rare.

The importance of the occurrence of cranial-nerve palsies in the early course of syphilis does not lie in their own seriousness, since they usually clear with treatment and often even without treatment, but in that they are often a forewarning of the later development of a more serious involvement of the nervous system.

The development of optic-nerve atrophy in the course of syphilis of the central nervous system is the most perplexing problem that confronts the syphilologist. The pathogenesis of the atrophy is poorly understood and all of our therapeutic armamentaria are aften of no avail.

Derby in 1902 estimated that syphilis was the cause of the atrophy in 7.3 percent of 108 cases of optic-nerve atrophy. Woods found that syphilis was the cause of the atrophy in 17 percent of 137 cases admitted to the Wilmer Institute.

It is well known that it is difficult to estimate the severity of the visual loss by the appearance of the optic disc. Relatively good vision may be preserved,

although the ophthalmoscopic picture is that of a pale, or white nerve head. The defects in the fields of vision are quite varied. Often there is a constriction of the peripheral fields with enlargement of the blind spot. Homonymous hemianopsia is rare, but binasal contraction of the fields (so-called binasal hemianopsia) is sometimes seen. The pathological picture is a combination of an inflammatory reaction and degenerative changes in the myelin sheaths and axis-cylinders. The degree of degeneration and inflammation need not be parallel. Spirochetes are rarely found. These facts make any hypothesis as to the pathogenesis difficult to prove.

Every known method of combating the disease syphilis has been used in the treatment of the optic atrophy, including intravenous and intramuscular injections, intralumbar, intracisternal, and intraventricular serum injections, fever, and even tryparsamide. The majority of the reports in the literature suffer from various defects, chief among which is a lack of coöperation between syphilologist and ophthalmologist with resulting failure of figures as to visual acuity and accurate visual-field studies before and after treatment.

Any discussion of the ocular damage done by syphilis is incomplete without a brief mention of that produced by the physician himself in his attempts at therapy, namely, tryparsamide amblyopia. The reality of such cases cannot be doubted by anyone who has encountered the sudden onset of blindness in a patient he is treating for neurosyphilis with tryparsamide.

It is estimated that 15 percent of patients treated with tryparsamide develop symptoms of damage to the optic nerve. In over two thirds of such patients such symptoms are of minor significance as regards serious damage, and consist of dazzling flashes of light or a shimmering sensation before the eyes, developing 6 to 24 hours after an injection. The importance of these symptoms lies in that they are a warning of more serious damage if treatment is continued. More serious damage to the optic nerve is indicated by the sudden occurrence of amblyopia. Ophthalmoscopic studies in the acute stage are negative, but testing of the visual field will show an extreme degree of contraction of the peripheral fields. Optic atrophy develops months later. Prevention of ocular damage by tryparsamide is accomplished by a careful testing of visual acuity and fields before starting treatment and the careful observation for the early symptoms of optic-nerve damage. The drug should not be given to patients with preëxisting diseases of the optic nerve, and its use should be discontinued or continued only with extreme caution in patients who develop ocular symptoms after an injection.

Discussion. Dr. Robert Schwab said Dr. Merritt had contributed some important, original work on the Argyll Roberston pupil. Dr. Schwab said he would like to remind the Society of the differential diagnosis between this pupil and the myotonic pupil of Adie's syndrome. In the typical Argyll Robertson pupil due to syphilis there is no light reaction whatsoever. The pupil is small and irregular, the accommodation reflex is brisk, and the patient may have a lost ankle or knee jerk usually with some involvement of sensation. In Adie's syndrome the pupil will react to light after a delay, contracting sluggishly and then expanding again slowly. The accommodation reflex is also sluggish. The pupil is usually regular, and is usually involved on only one side with a perfectly normal pupil on the other. In Argyll Robertson pupils the involvement is frequently bilateral. Whenever these pupils are en-

countered, a Wassermann test and lumbar puncture will finally determine the diagnosis. It is also to be noted that frequently in Adie's syndrome loss of knee or ankle jerk is common as in tabes but sensation is intact.

In the differential diagnosis between ptosis due to syphilis and ptosis due to myasthenia gravis, which is of particular interest in the neurological clinic, a new test has been worked out. Dr. Schwab said that ptosis due to syphilis will not respond at all to an injection of prostigmin, whereas ptosis due to myasthenia gravis will disappear completely when prostigmin (3 c.c.) is injected intramuscularly.

Virgil G. Casten.
Recorder.

SAINT LOUIS OPHTHALMIC SOCIETY

February 24, 1939

Dr. B. Y. Alvis, *president*

Dystrophia adiposa corneae

Dr. Howard C. Knapp read a paper on this subject, published in this Journal (1939, v. 22, p. 1239).

Discussion. Dr. E. C. Spitze said he first saw this patient in June, 1929, when she was 25 years of age and doing laundry work. She stated that she had had an attack of severe "sore eyes" when six years of age and that her eyes had been "weak" ever since. She also stated that five months ago, a white spot appeared on the left eye, that two weeks ago both eyes had become inflamed, and a spot appeared on the right eye. Examination showed in the right eye a small white dense area in the lower nasal quadrant of the cornea, about 2 mm. in diameter, with numerous blood vessels extending to it. In the left eye there was a similar mass in the lower portion, 5 mm. in diameter. These masses were chalky white, dense, the margins sharply outlined, and with unbroken surfaces. There was not the faintest infiltration of the cornea beyond the borders of the masses. They resembled somewhat the salmon patch of interstitial keratitis, yet the appearance was not the same as seen in that disease. There was some congestion of the bulbar and palpebral conjunctiva. Vision, in the right eye was 20/60 corrected to 20/30; in the left eye, 20/100.

The pupils were small and did not react. They dilated to 5 mm. with atropine. The patient had been married at the age of 19 years. Her first pregnancy terminated in a miscarriage at two months. She had never been pregnant since. There were numerous small soft tumor masses in the skin all over her body, ranging in size from 2 mm. to 1 cm. in diameter. She stated that she had always had them. A blood-Wassermann reaction was negative.

Because of the peculiar appearance of these masses in the corneae together with the negative Wassermann reaction she was referred to Dr. M. H. Post, who suggested that the corneal lesions might be tubercular. He advised treatment with the Shahan thermophore. One application was accordingly made to the larger mass in the left eye for one-and-one-half minutes at 158°F. There was no change in the appearance of the mass. Under atropine and metaphen medication the inflammation gradually subsided. Ten days later the patient disappeared and was not seen again until July, 1935, six years after the first examination.

At this time the right eye was inflamed. It was treated for three days. She returned two weeks later with an acute purulent conjunctivitis in both eyes. The masses in the corneae were now twice the size they were when first seen. In the left eye there was a small ulcer of the cornea with indurated edges at each end of the mass. Again a blood Wassermann

was negative. The ulcers were cauterized and the eye treated with silver, atropine, and metaphen. Improvement was very slow, but at the end of two months the eyes were in good condition except for the masses in the corneae, which remained the same as before the treatment. She was not seen again until her presentation before the Society.

Fuchs, in his text book, states "The dystrophies are chronic disturbances of nutrition of the cornea which lead in the course of years to a constantly increasing opacity. The dystrophies are distinguished from the inflammations of the cornea by (1) the absence of external symptoms of inflammation, such as injection, pain, and so forth; (2) the constantly progressive course; (3) the anatomic findings, which show degenerative processes (fatty degeneration)—in many cases the epithelium over the opacity is dull, an indication that a progressive process is present. In many cases the cause of the dystrophy remains unknown."

Dr. Max Jacobs said that in the concluding sentences of the essayist's paper he made the statement that certain investigators had found that even in high cholesteremia, cholesterin was not deposited in the cornea. He would like to ask the essayist if he can recall whether this work was done recently or at a distant date. His reason for asking is this: Rohrschneider wrote a number of papers several years ago and showed by animal experimentation that by feeding laboratory animals a high diet of cholesterin he would get deposits simulating arcus senilis. Later, reducing the cholesterin content of the diet, these deposits disappeared or faded out considerably. Rohrschneider, at that time, offered this as the explanation of arcus juvenilis found in young people. The fact that we are dealing with end-products of cholesterin makes it pertinent to find out whether these data antedate or follow the work of Rohrschneider.

Dr. Howard C. Knapp in conclusion said this investigation was completed in 1932. He believes that the deposition of cholesterin in the cornea accomplished in experimental animals is not accompanied by disintegration of tissue and the cell structure is not damaged. In adipose degeneration, the cell is functionally damaged and would be incapable of returning to its normal condition if the fat were removed.

Chemical destruction of the lacrimal sac

Dr. W. E. Shahan read a paper on this subject.

Discussion. Dr. J. M. Keller stated that Dr. Shahan is to be complimented on having conceived the idea and having developed a technique by which the chemical destruction of the tear sac can be accomplished through the natural route; namely, through the canaliculi.

Heretofore, advocates of the chemical destruction of the tear sac reached the sac through a skin incision. Dr. Shahan enters the sac through the canaliculi with an especially constructed medicine dropper and then deposits his cauterizing agent. The operation is bloodless, reduces the time required for healing, leaves no visible scar, and can easily be done in the office.

Although the chemical destruction of the mucous membrane of the lacrimal sac is one of the oldest methods used in the treatment of chronic dacryocystitis, Dr. Shahan's method of approach and technique are new. A drawback of the chemical-cauterization method is that one is never quite certain whether or not all of the mucous membrane has been destroyed. A small remnant of remaining mucous membrane is sufficient to cause all the symptoms of dacryocystitis to return. Nevertheless, many excellent results by

this method have been reported. The only absolutely positive elimination of this source of infection is the extirpation of the tear sac *in toto*. If this is accomplished there is no remaining fragment of mucous membrane to cause further trouble. If, however, extirpation of the sac is impossible or impractical, as in Dr. Shahan's case, the chemical destruction is very useful. Extirpation should never be attempted during an acute inflammation of the tear sac because of the possibility of spreading the infection, the technical impossibility of dissecting out the sac *in toto*, and the danger of thrombophlebitis of the orbit with a fatal result.

During the past 25 years dacryocystorhinostomy has gained considerable ground. This operation, while it deals more conservatively with the tear passages and endeavors to restore, as nearly as possible, the physiologic conditions, is technically difficult and more trying, especially on frail and infirm patients.

Dr. W. F. Hardy said there were several points in Dr. Shahan's paper he would like to discuss. Dr. Shahan speaks about preventing the regurgitation of trichloracetic acid along the syringe needle end. This is necessary if a burn of the conjunctiva is to be avoided. In syringing with the same type of syringe it is not uncommon to have fluids, either boric-acid solution or saline solution, regurgitate into the conjunctival sac either along the needle or through the upper punctum. Another point is that many lacrimal sacs are friable and allow simple fluids to break through and infiltrate the subcutaneous tissues in syringing. Hence the use of all silver solutions is interdicted. Furthermore in many cases of old dacryocystitis the sacs are dilated and have lost all contractility so that it might be difficult to get to all parts of the sac and destroy the mucous membrane with trichloracetic acid.

For 15 years or more he has been performing the Gifford operation by thoroughly exposing the sac, incising it, and destroying the mucous membrane in its entirety. It has been most successful not only as a preliminary to cataract operation but in ordinary cases of chronic dacryocystitis. As far as the resulting scar is concerned it is minimal. It has been his custom to use no drain, no pack, and no suture. In a few days the skin incision becomes sealed, the wound heals beautifully, and in a few months it is difficult to find the scar.

Dr. John Green said if he understood Dr. Shahan correctly several applications of the cauterant may be necessary before the mucosa is destroyed. Despite precautions there is some danger that the mucosa of the conjunctival sac may be damaged. Should there be a weakness in the wall of the lacrimal sac or an actual dehiscence there is danger of the cauterant's getting into the tissues outside the sac.

Given a hypopyon ulcer complicated by dacryocystitis it is vitally important to banish immediately the source of infection. Under these circumstances he prefers to open into the sac and to cauterize the mucosa with trichloracetic acid, following the technique of Harold Gifford.

Dr. W. E. Shahan, in conclusion, said that in chemical destruction of the lacrimal sac the solution is introduced through the dilated canaliculus or through a previously existing fistula by means of a platinum-iridium needle fused in the end of a glass dropper. The sac is first anesthetized by injecting 5-percent cocaine in 1:4000 adrenalin into the sac through a small needle fused in the tip of an ordinary rubber-capped eye dropper. A dropper with a larger needle is then introduced through the canaliculus and the sac thoroughly washed out by

repeatedly injecting and expressing a quantity of normal salt solution. This is done by alternately squeezing on the rubber bulb of the syringe and on the distended lacrimal sac. After the sac is thoroughly cleansed a little more anesthetic is injected into the sac. After a few minutes this is pressed out and the sac left clean and flat.

The conjunctival sac is then filled with vaseline, and cotton is banked about the punctum to keep any of the corrosive solution from spilling into the conjunctival sac. The syringe with the larger needle is now filled about one-fourth full of a saturated solution of salicylic acid in 95-percent alcohol. This needle is quickly introduced into the sac through the canaliculus and the solution regurgitated back and forth from the sac to the syringe a number of times. Finally the sac is pressed flat and free from solution and the syringe is withdrawn. The solution coagulates blood and pus quickly so that if a drop gets into the syringe it will block it and prevent its functioning. When this occurs, the rubber bulb is removed from the syringe, the solution is poured out and the clot burned out of the needle over a Bunsen burner.

The corrosive action of this solution does not penetrate deeply, so that several treatments may be necessary before the sac walls have completely grown together. Subsequent reaction is rather mild. In one particularly stubborn case with chronic fistulae a saturated aqueous solution of trichloracetic acid (suggested by Gifford's technique) was used in addition to the above solution. The patient was an elderly woman in whom any violent measure was contraindicated and who was practically blind from cataract. After destruction of the sac and canaliculi and after bacteriological investigation in the hospital, she was successfully operated on and has had excellent vision for the remaining years of her life.

Ocular tularemia

Dr. Carl Gissy gave case reports upon this subject.

Discussion. Dr. H. N. Fisher stated that he would like to draw attention to the use of metaphen (1:1000) intravenously in cases of tularemia. The literature does not contain any mention of its use in cases of this kind, but it has been used in a large number of cases over a period of several years at the Olney Sanitarium at Olney, Illinois, and so far there have been no failures. It was first used with success on one of the younger members of the staff after other recommended remedies had failed, and since then it has been the routine method of treatment there. The average dose for adults is 10 c.c. intravenously daily for several days until improvement is noted, then the interval between injections is lengthened as indicated. Since joining the staff he recalled a boy about 18 years of age who came in with what at first appeared to be a severe case of simple conjunctivitis with enlargement of the preauricular gland. When he returned on the third day he was much worse after using optochin hydrochloride in the eye every two hours. His temperature was high, he looked sick, his preauricular gland was much larger, and the whole chain of glands in the anterior portion of his neck was enlarged. He was given 10 c.c. of metaphen (1:1000) intravenously and showed a decided improvement the next day. Injections were repeated at intervals of one to three days, and the condition cleared up entirely at the end of the second week with no complications. The results of the whole series of cases will be reported later in the literature by a member of the staff.

Adolph Lange,
Editor.

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GLAUCOMA

The highly specialized nutrition of the eye provides for the transparency of the refractive media and the maintenance of its optical proportions. These conditions are totally different from what is achieved by the metabolism of any other part of the body. The history of glaucoma shows its complete confusion with cataract, atrophy of the optic nerve, and other conditions with which its connection was entirely accidental. Priestley Smith opened the door to the understanding of this disease when he defined glaucoma as increased intraocular tension, plus the causes and effects of such increase. But the general investigations of metabolism and nutrition, turned in other directions, threw very little light upon physiological intraocular tension, or the mechanism that maintained it.

Mackenzie had recognized the importance of ocular hypertension; but nothing was done for it until Graefe noticed that iridectomy sometimes lowered the intraocular pressure. It was soon accepted as a proper treatment for glaucoma. Not the kind of iridectomy used to make an artificial pupil, or as part of a cataract extraction; but a wider iridectomy, removing the iris all the way to its attachment to the sclera. For acute cases with little inflammation, it is still recognized as good treatment. But for many of the cases it proved ineffective. Hancock made a radial section through

the ciliary body. This has also been done by a few operators, with good success. Posterior sclerotomy has been used to reduce the tension temporarily, before performing another operation for permanent effect. Lagrange, with iridectomy, cut off a piece of the sclera. Heine, through an incision made back of the ciliary body, did cyclodialysis; separating the choroid and ciliary body from the sclera. Herbert's flap operation separated the sclera from the iris back of the limbus. Argyll Robertson suggested trephining the sclera to secure permanent drainage. Elliot trephined at the sclerocorneal junction with, or without, removal of a small piece of iris. This, extensively practiced in Egypt and India, was found to relieve the chronic cases there encountered; especially if it caused a bulging, or cystoid cicatrix. Iris-inclusion operations, in which portions of the iris were drawn into the corneoscleral incision, and in Holth's operation into a trephine opening, have also relieved cases of chronic glaucoma, and have been resorted to as being easier and less dangerous than other glaucoma operations.

Each of these operations has been advanced and supported with theoretical discussions of the pathology of glaucoma; and the same is true of the latest procedure of Otto Barkan, opening the angle of the anterior chamber and Schlemm's canal by an incision, under direct observation with a modified corneal microscope. All the operations, by allowing direct escape of fluid from the interior of the eyeball, have produced an immediate decrease of intraocular pressure. In some cases the effect has been permanent; so that each operation is supported by a certain number of cures. But in other cases that seemed to be favorable, the relief has not been permanent; and in spite of all treatment the eyes have gone on to permanent blindness. It is evident that our knowledge of glaucoma is incomplete. Some factor in the pathological hardening of the eyeball has been overlooked.

On this account, the study of glaucoma by Luedde (page 388), calling attention to the probable function of the capillary epithelium of the smaller blood vessels in the eye, and the probable escape of fluid through the permeability of the peripheral cornea, as observed by Ridley, seems to fill up a waiting gap in our understanding of glaucoma. With this we may well consider the relief of glaucoma reported by Miller, Luedde, and others, in a few cases. It points to the possibility of a tendency to glaucoma that may be removed possibly by administration of splenic extract, or other means. The problem of glaucoma has often ended in what appeared to be an impasse; but these recent suggestions may open a new field in ophthalmic therapeutics.

EDWARD JACKSON.

RETINAL CAPILLAROPATHY

The invention of the ophthalmoscope did more than expose to view during life the vital structure of the organ of vision. It placed before our eyes, in the living subject, a portion of the brain itself, with the ramifications of its blood supply in health and disease. For the retina is a highly specialized outgrowth of the brain, subject in large part to the same vascular disorders, the same degenerative changes, as occur in the brain itself. Many lessons as to cerebral pathology have been deduced from the pictures disclosed by the ophthalmoscope; and the story is still being unfolded.

For a long time the chief emphasis in regard to vascular disease in the retina was upon the larger vessels. But from retinal arteriosclerosis we have now progressed to thinking more seriously of

retinal arteriolar sclerosis, disease of the finest branches of the central artery. Here again, what we learn about arterial changes in the retina may be applied extensively to the phenomena of the cerebrum.

About the capillaries, in any part of the body, less has been known. They are most intimately related to nutritional activity. Any tendency to disease which they manifest must be of immense importance everywhere, but particularly so in the brain and in the retina, where we are dealing with "end arteries," vessels whose lesions are superlatively essential because of the absence of anastomoses and collateral circulation.

The real capillaries of the retina (as contrasted with minute arterioles) are made up of elongated endothelial cells. They are of such narrow caliber that the blood cells generally pass through them in a single column, usually undergoing some change of shape.

In spite of the relatively high magnification under which retinal details are viewed by the examiner through the optical system of the eye, many finer details of circulation and pathology are beyond our reach in the living subject, and are likely to remain so. Bailliart, who is so justly distinguished for his researches and writings concerning retinal circulation, published three years ago a fascinating article on functional study of the smallest vessels and the capillaries of the retina (Bulletin de la Société d'Ophtalmologie de Paris, 1937, April, page 204).

Using Fortin's special entoscopic technique (Archives d'Ophtalmologie, volume 27, page 397; and Archivos de Oftalmologia de Buenos Aires, 1937, volume 12, July, page 456), Bonnet as quoted by Bailliart is said to have assembled statistics as to the relative frequency with which the entoptic sensations associated with movement of blood cells were obtained at different ages and in normal and hypertensive subjects respectively. Among one hundred persons between the ages of twenty and forty years, vision of the blood cells with perception of rhythmic movement corresponding to the arterial pulse was recorded by eighty-three. Among one hundred persons of sixty years or more, only thirty were able to record the sensation, and above the age of seventy-five years the experience was exceptional.

According to Bailliart these experiments confirm the opinion that the capillary network in general and that of the retina in particular are less permeable in old age. Further studies by Bonnet upon hypertensive subjects gave analogous results. In moderate hypertension (brachial diastolic pressure of above 120 millimeters) the endoscopic evidence of capillary insufficiency occurred in seventy-two percent of the cases; among more marked cases of hypertension it was found in eighty-four percent. Herein lies the interpretation of many defects of senility, and especially of degeneration at the macula.

Bailliart has demonstrated that from the primary branches of the central retinal artery to the primary branches of the central vein the pressure in a normal subject falls from thirty-five millimeters to twenty millimeters. Thus, a reduction of fifteen millimeters has sufficed to maintain the current through the capillary network, with its nutritional implications. When the arterial pressure is lower, the force available to keep the capillary network open is weaker. The least spasm will then be enough to stop the capillary circulation.

When, in connection with an examination for glasses, or in making a mere visual record, we find it impossible to demonstrate a normal visual acuity, we can only assume without direct proof that some invisible circulatory and tissue

changes are at fault. Such invisible, or very vague, fundus changes are common in older patients. When definite changes are found they frequently take the form of minute yellowish or whitish dots in or near the posterior pole of the eye. These lie in the retina particularly along the small blood vessels. They may be regarded as pointing to inadequate circulation of blood in the retinal capillaries, with a consequent deficiency of nutrition in the retinal elements.

Graefe called attention to such dots as early as 1866. The English ophthalmologist Gunn described them in an early volume of the Reports of the Royal London Ophthalmic Hospital, and they have very commonly been referred to as "Gunn's dots," although curiously enough that writer himself preferred to speak of them as "Crick's dots." Horniker called the condition "central angioneurotic retinitis," Bailliart and Goldzieher used the name "capillaritis," while Cattaneo preferred the somewhat meaningless term "retinosis."

In an excellent review of the literature of the subject, accompanied by several case reports, Avizonis (Ophthalmologica, 1939, volume 98, November, page 129) suggests that since the dots cannot be supposed to arise from an inflammation, neither "retinitis" nor "capillaritis" is logically applicable. In some cases, Avizonis argues, the basis is organic or plastic closure (partial or complete) of the capillaries and precapillaries, producing local disturbance of the circulation. For these reasons Avizonis thinks the expression "retinal capillaropathy" more suitable than those previously put forward.

The dots, which Bailliart considers analogous to the discrete lesions making up a focus of circinate retinitis, are interpreted by Avizonis as representing very small hollow spaces in the retina, produced by softening of the retina after closure of the capillary vessels. Avizonis describes a case in which indirect illumination of the retina with the electric ophthalmoscope showed many minute depressions or excavations, although by direct illumination they had the appearance of yellowish-white dots.

On the basis of histologic and clinical investigations, Mawas, Morax, and Bailliart maintain that in persons with this retinal condition a similar process goes on in the brain, where from the same cause there arise small foci of softening which give rise to the cerebral lesions described by Marie and others. Herein would lie the anatomic foundation of such degenerative manifestations as changes in character, failure of memory, mild attacks of dizziness, and difficulties in hearing. All these phenomena of senescence are explicable as resulting from small cavities in the brain. Various authors have convinced themselves that small foci of cerebral softening may be due to spasm of the capillaries of the brain.

Why, it may be asked, does not the same process affect the capillary vessels of the choroid? But the choriocapillaris is rich in the anastomoses which are lacking in the brain and retina, so that circulatory disturbances in the choroidal network are less destructive.

Prophylactically and therapeutically, Avizonis regards the early recognition of retinal capillaropathy as highly important. It is widely considered that vascular spasm may lead in time to anatomic changes in the vessel walls. Thus it becomes necessary to counteract, as far as possible, such causes of spasm as toxins, nicotine, renal insufficiency, qualitative and quantitative defects in the supply of hormones, and sudden marked changes in blood pressure.

Although recognizing that senile de-

generation of the macula is incurable, Avizonis is optimistic enough to believe that its development may be retarded therapeutically after the preliminary changes have been recognized. The most important early diagnostic sign is the finding of yellowish-white points in the macular area or its vicinity. In the presence of these and other premonitory vascular changes he administers acetylcholine, in daily intramuscular injections of 0.1 gram. In several cases, material improvement in vision is said to have occurred after the third or fourth injection. Bailliart uses the same drug subcutaneously, in doses of 0.1 to 0.2 gram.

Consideration of these degenerative vascular changes, quite commonly regarded as more or less familial or hereditary in character, raises the interesting question to what extent it is possible to influence in any lasting or substantial degree those processes which are rather involutional than due to actual disease.

W. H. Crisp.

BOOK NOTICES

LEHRBUCH DER AUGENHEILKUNDE (Textbook of ophthalmology). By Dr. Ernst Fuchs; 16th enlarged and improved edition, revised by Dr. Adalbert Fuchs, Associate Professor of Ophthalmology in the University of Vienna. 917 pages, 362 reproductions (many of them colored), and 5 color plates. Vienna, Franz Deuticke, 1939. Price, paper covers, 27 marks, bound, 30 marks.

In 1881 an assistant in Arlt's Clinic at the University of Vienna was seriously considering migration to South America. He had already taken passage to a South American port (according to the late Adolph Barkan) but the assistant received an invitation to assume the Professorship of Ophthalmology at the University of Liége. After four years in that position he was appointed Chief of the Second Eye Clinic in Vienna. The young assistant was Ernst Fuchs, who in course of time became perhaps more widely known to the medical world, and especially to ophthalmologists, than has been any other ophthalmologist past or present. During his assistantship to Arlt he had given the first medical course in Vienna in the English language. Many years later, upon a suggestion by Edward Jackson, he established the first three-month ophthalmologic course in English at the same university.

In 1889 Ernst Fuchs published the first edition of his famous "Textbook on ophthalmology" whose genesis he explained in the following humorous phraseology: "In German universities . . . industrious students take the trouble of putting their teachers' words on paper. This . . . in my course . . . happens not at my desire but . . . against. . . . It disturbs the student's attention to what he sees, and the rereading causes great waste of effort and valuable time. . . . If students so warmly desire to possess their own teacher's words in black and white, why should not one accommodate this desire as far as possible."

The work thus modestly introduced to its readers now appears in a sixteenth edition, revised by the author's son Adalbert. The latter reminds us of the fact that the description by his father of external eye diseases was so classical "that in many parts of the world his textbook was characterized as the 'ophthalmologist's Bible.'" Beside translation into several other foreign languages the volume has appeared in nine English editions.

In the present German edition the color reproductions of pathologic preparations are taken from Adalbert Fuchs's "Atlas

of histopathology." Recent advances are mentioned, although some of them with extreme brevity. The book frankly does not profess to describe surgical procedures. But as a description of disease conditions found in the eye and its adnexa it still remains in many respects the world's outstanding authority. The printing and editing of the new edition are beyond reproach.

W. H. Crisp.

TRAITÉ D'OPHTALMOLOGIE. Published under the auspices of the Société Française D'Ophtalmologie. Edited by Bailliart, Coutela, Onfray, Redslob, and Velter. Paris, Masson et Cie, 1939, volumes VII and VIII

With the appearance of volumes VII and VIII, almost on schedule in spite of war, the series comes to an end. The last two volumes continue the high standard established by the others. Hearty congratulations and words of praise are due the editors, contributors, and publishers for their splendid achievement. By the same token congratulations are due the French reading ophthalmologist for the treat in store for him.

Volume VII (938 pages, 414 figures, 31 colored plates) continues ocular neurology where volume VI left off. Professor Velter contributes two important chapters to this part of the volume. One is oculomotor and pupillary disturbances and the other, symptomatology of ocular diseases in the affections of the central nervous system. Both are well written and illustrated. René Onfray, who is the general secretary of the French Ophthalmological Society, thoroughly covers the subject of strabismus. Professor Coppez clarifies the usually difficult subject of nystagmus in a short but adequate chapter. Cerise and Thurel write on ocular diseases produced by lesions of the trigeminal nerve. Vincent and Puech analyze the chiasmal syndrome from their authoritative neurosurgical point of view. De Martel and Guillaume describe the principles of diagnosis and treatment of intracranial hypertension and cerebral tumors as well as ventriculography.

The remaining 550 pages of the volume are devoted to ocular surgery in all of its aspects. Much of its value is due to the many beautiful colored plates, as well as black and white figures, adequately illustrating the different steps and procedures of every conceivable ophthalmic operation. The contributors are Saint-Martin, Duverger, Velter, Merigot, Treigny, Van Lint, Veil, Dollfus, Onfray, Carlotti, Terrien, Bruneau, and Charpentier. Those familiar with French ophthalmology will recognize the authority of these names of master surgeons.

Volume VIII (1,000 pages, 164 figures, 2 colored plates). A third of the volume is devoted to medical therapeutics. J. Plicque writes three valuable chapters, the technique of common local medications, biologic therapeutics, and general indications and therapeutic synthesis. Margerin's chapter on medicinal therapeutics is particularly noteworthy. His formulary is complete, sound, and profitable. Professor Reiss of Strasbourg writes on physical agents, excluding radiotherapy, which is described by Gunsett. Liégard covers the subject of eyeglasses in an interesting fashion, and Haas that of contact glasses.

The middle section of the volume is given over to the subject of industrial hygiene. The contributors are Pesme, Bonnardel, Chappé, Cantonnet, Monthus, and Coutela, whose book "L'oeil et les maladies professionelles" was recently reviewed in these columns. The section on legal medicine, also by Coutela, is of interest to the American reader because

of the different way this matter is handled by the French.

Professor Toulant of Algiers devotes 154 pages to tropical ophthalmology, which is well done. One wishes, however, there had been more of the striking colored plates or autochromes by Dr. Morard to illustrate this section. Professors Robin and Brion, veterinarians, collaborate on a chapter, unique in ophthalmic systems, on comparative ophthalmology This should be a useful work of reference to ophthalmologists doing animal experimentation. Cosse and Caillaud's chapter on medical ethics and ophthalmology is an important and fitting end to a magnificent piece of work. An alphabetic index of the entire set of eight volumes is appended. It would have been more useful, I think, had it been bound separately.

Derrick Vail.

BULLETINS ET MÉMOIRES DE LA SOCIÉTÉ FRANÇAISE D'OPHTALMOLOGIE. 403 pages. Paris, Masson et Cie, 1939, volume 52.

This annual volume presents the individual reports made during the meetings of May 9 to 11, 1939, and comprises 37 communications. The subject matter covers a diversified assortment and does not lend itself to facile or coherent review. The interest in the different reports will, therefore, fluctuate with the personal interests of the different readers.

The first 47 pages of the text are devoted to the discussional comments of several members to a report made before the society by Dr. Coutela on professional and industrial diseases of the eye. Unfortunately, the report itself is not given either *in toto* or in abstract, so that it is difficult to appraise fairly the different comments.

Several papers are given over to anaphylactic conditions of the eye: Drs. Sédan and Koutseff illustrating the effect of ovarian hormones on ocular allergy; Dr. Nectoux demonstrating by experimental procedure the increased ocular tension accompanying anaphylactic reactions of the rabbit's eye. Cholesterin infiltration of the cornea and its treatment by cholesterin solvents are discussed by Genet and Martin.

Predicating trachoma as a lymphoidal disease, Dejean and Artières give a theoretic consideration of benzene and its derivatives in the treatment of trachoma because of the use of this chemical in leukemic hypertrophy and lymphoid adenitis. They have actually treated 15 trachomatous patients with septicine (a sulfon, as well as benzene compound). They found marked improvement in 12 patients, slight improvement in 2, and no change in 1.

A method of diagnosis for leprous keratitis is suggested by lifting off a corneal nodule, and staining the crushed contents by the usual acid-fast stains. There is an interesting paper on ocular pemphigus by Kapuscinski, in which the concept is postulated that pemphigus is primarily a degeneration of the conjunctival and corneal epithelium and that inflammatory infiltration is a secondary phase.

As was true last year, there is more or less a "symposium" on cataracts, with reports on extraction. In one paper, the treatment of senile cataract with ascorbic acid is reported, apparently with encouraging results.

It would be impossible in a review of this kind to review in great detail all the papers. It should be said, however, that the volume maintains its usual good standard of reporting and publication.

L. A. Julianelle.

CORRESPONDENCE

Calcium equivalents of preparations

Editor,
American Journal of Ophthalmology

I have received several communications about the vitamin-D–complex therapy in keratoconus. In most of these letters I have been asked what calcium preparation can be substituted for Mead's Mineral Mixture ♯85, which product the Mead Johnson Company has discontinued. I have not used any other calcium compound, but it is my impression that any popular calcium salt may be used, though calcium disulphate seems to be preferred. It is probable that frequently the substituted products prescribed contain an insufficient supply of calcium. Therefore, I thought it advisable to publish the calcium equivalents of these several well-known preparations. One gram of calcium will be contained in 3.57 grams of Mead's Mineral Mixture ♯85, 4.30 grams of Dicalcium phosphate, 7.70 grams of Calcium lactate, 11.21 grams of Calcium gluconate.

Another way of expressing the same thing would be to say that 1 gram of Mineral Mixture ♯85 contains 0.28 gm. calcium, 1 gram of Dicalcium phosphate contains 0.2324 gm. calcium, 1 gram of Calcium lactate contains 0.1298 gm. calcium, 1 gram of Calcium gluconate contains 0.0892 gm. calcium.

For the vitamin D, may I suggest at least 60 drops of viosterol.

I hope that this information will be of some help to those desiring to avail themselves of this therapy.

(Signed) Arthur A. Knapp.

ABSTRACT DEPARTMENT

EDITED BY DR. WILLIAM H. CRISP
ASSISTED BY DR. GEORGE A. FILMER

Abstracts are classified under the divisions listed below, which broadly correspond to those formerly used in the Ophthalmic Year Book. It must be remembered that any given paper may belong to several divisions of ophthalmology, although here it is only mentioned in one. Not all of the headings will necessarily be found in any one issue of the Journal.

CLASSIFICATION

1. General methods of diagnosis
2. Therapeutics and operations
3. Physiologic optics, refraction, and color vision
4. Ocular movements
5. Conjunctiva
6. Cornea and sclera
7. Uveal tract, sympathetic disease, and aqueous humor
8. Glaucoma and ocular tension
9. Crystalline lens
10. Retina and vitreous
11. Optic nerve and toxic amblyopias
12. Visual tracts and centers
13. Eyeball and orbit
14. Eyelids and lacrimal apparatus
15. Tumors
16. Injuries
17. Systemic diseases and parasites
18. Hygiene, sociology, education, and history
19. Anatomy, embryology, and comparative ophthalmology

1

GENERAL METHODS OF DIAGNOSIS

Bair, H. L., and Harley, R. D. **Midline notching in the normal field of vision.** Amer. Jour. Ophth., 1940, v. 23, Feb., pp. 183-185.

Comberg. **Testing light sense with the ophthalmoscope in unilateral eye affections.** Deutsche Ophth. Gesell., 1938; in Klin. M. f. Augenh., 1938, v. 101, July, p. 110.

Comparison of this function in the affected eye with that of the normal eye is of practical importance in most eye injuries and in many cases of nontraumatic pathology. The modern electric ophthalmoscope is quite suitable if its illumination can be focused uniformly on a sharply defined field. The eyes are tested separately and alternately, with the lids closed so as to act as a screen which diffuses the light uniformly. With this procedure there is sufficient sensitivity to differentiate slight changes in intensity of illumination; an alteration of one fifth in distance or one third in illumination being appreciated at once even by inexperienced or unintelligent subjects. The test is simple, rapid, and sufficiently accurate for all clinical conditions. Tests with an adaptometer do not usually improve on the interpretative value of the findings.

Percy Fridenberg.

Evans, J. N. **Classic characteristics of defects of the visual field.** Arch. of Ophth., 1939, v. 22, Sept., pp. 410-431.

Two types of visual-field defects, neurogenic and angiogenic, are distinguished by the nature of their origin. Neurogenic defects are those that arise in association with lesions of the nerve-fiber bundles of the retina, and angiogenic defects are those that arise from vascular and nutritional disturbances. A typical defect of fiber-bundle origin is wedge-shaped with its apex pointing to the fixation point, there are no pronounced fluctuations at its borders, and in arrangement it corresponds to the fiber bundle of the optic pathway. A typical defect of vascular origin is also wedge-shaped, but its apex points

toward the blindspot, there are transient fluctuations at its borders, and its arrangement corresponds to the patterns of the retinal-vessel tree. Distinction between these two types of defects is of special value in intracranial lesions. A better interpretation of clinical material can be obtained by combination of neuroscotometry and angioscotometry. J. Hewitt Judd.

Franceschetti, A., and Borsotti, I. **The importance of the pupillographic method of Löwenstein in ascertaining physiologic and pathologic pupillary reactions.** Riv. Oto-Neuro-Oft., 1939, v. 16, May-June, pp. 183-213.

The synchronous cinematographic registration of the two pupils by the Löwenstein pupillograph is described in detail. By this method a film, which has been taken at a speed of ten images per second, is projected on a screen, and the pupillary diameters are measured image by image. The values are reported in diagrams. The direct and consensual reactions to light and obscuration, and the convergence-accommodative and pharmacodynamic reactions are tested. The practical importance of pupillography consists in getting evidence of changes in the reflexes and localizing the lesions that provoke these changes in the nervous system. It also aids in recognizing progression or regression of the pathologic processes, and in analyzing these factors before neurologic or other examination becomes valuable, especially as regards the early diagnosis of luetic and metaluetic affections. Nine pupillograms are reported from luetic patients, one from an anisocoric patient, two showing the tonoaptic reaction in diencephalic lesions, two of Adie's syndrome, and two showing the effect of sympathicomimetic substances. (Bibliography, 34 figures.) M. Lombardo.

Graff. **A new reflex-free ophthalmoscope.** Deutsche Ophth. Gesell., 1938; in Klin. M. f. Augenh., 1938, v. 101, July, p. 109.

The form and measurements of this instrument correspond to those of the usual electric ophthalmoscope but it is strictly free from reflexes. Using the indirect method, reflexes from the accessory lens can easily be eliminated. Percy Fridenberg.

Kalashnikov, B. P. **Ocular ergography for determination of intestinal autointoxication.** Viestnik Opht., 1939, v. 15, pt. 2, p. 60.

The author believes that ergographic study of accommodation after internal administration of atropine may indicate adequacy of liver function in elimination of the drug. When the barrier function of the liver relative to toxins is impaired, atropine invades the circulation, and the ergograph demonstrates its effect on accommodation. Intestinal toxins may also act on accommodation under similar circumstances. Essential weakness of accommodation may therefore be caused in some cases by the action of intestinal toxins in the presence of hepatic insufficiency; in such cases ergography may be used as a quantitative indicator of liver function. Premature presbyopia may be the result of intestinal intoxication. The author calls attention to the possibility of regulating the adequacy of the liver function by diet. Ray K. Daily.

Lijo Pavia, J. **Tonoscopy and papilledema.** Rev. Oto-Neuro-Oft., 1939, v. 14, Jan., p. 7. (And see Amer. Jour. Ophth., 1940, v. 23, Jan., p. 107.)

The author cites instances to controvert the assertion of numerous authorities that retinal arterial hypertension either does not exist or is inconstant

when papilledema appears as a manifestation of increased intracranial pressure. His contention is that the retinal arterial pressure is increased in papilledema and may, therefore, be regarded as an important differential point in the diagnosis of incipient swelling of the nerve head. He discusses the technique and problems of venous tonoscopy, affirming that it gives direct information with respect to the pressure within the veins and venous sinuses of the cranial vault. The final conclusions are: (1) In the presence of papilledema, arterial or venous tonoscopy should be carried out with a dilated pupil. (2) If the central artery or its primary branches cannot be seen, redfree illumination should be utilized. (3) If, in spite of this, the arteries cannot be seen and pulsations cannot be produced in them, acetylcholine should be administered to eliminate the possibility of angiospasm. (4) If, on compressing the globe with the dynamometer, segmentation of the venous current is produced before the value of the maximal retinal arterial pressure is attained, it will be an indication that the arterial blood supply is deficient. (5) It is probable that in the latter case the nutrition of the retina will be seriously compromised if decompressive measures are not effected. (6) The facile occurrence of segmentation of the blood column is a valuable differential diagnostic sign between early papilledema and optic neuritis.

Edward P. Burch.

Posteli, T., and Seidenari, R. **Contribution to the clinical application of electroencephalography.** Riv. Oto-Neuro-Oft., 1939, v. 16, Jan.-Feb., pp. 1-14.

The authors employed electroencephalography in obtaining a registration of the cerebral waves in 32 normal persons and in five pathologic cases, of which two were of glaucoma and one of retinal detachment. The alpha waves which originate in the occipital region, the cortical site of vision, were studied in the eye cases. Special modifications of the tracing were noted on the side of the affected eye. In order to give a proper interpretation of findings and to distinguish the electric component of ocular origin in the electroencephalogram, the experiments were repeated on human and rabbit enucleated eyeballs. It was possible to note in the dying eye the gradual disappearance of the electric manifestations. (9 figures.) M. Lombardo.

Rosenthal, C. M. **Changes in angioscotomas associated with inhalation of oxygen.** Arch. of Ophth., 1939, v. 22, Sept., pp. 385-392.

By means of the Evans method of angioscotometry 15 subjects were examined before, during, and after inhalation of oxygen. It was found that a narrowing of the angioscotomas occurred during the period of inhalation and that after withdrawal of the oxygen there was a widening of the angioscotomas in excess of the amount shown by the first or control chart. It is thought that the narrowing is due to increase in the oxygen content of the retina rather than elsewhere in the visual pathway. The changes in the scotomas are clearly shown in charts.

J. Hewitt Judd.

Waugh, D. D. **An improved test object for perimetry.** Arch. of Ophth., 1939, v. 22, Sept., p. 450.

This consists of a glass-headed pin on the end of a holder, the pin being bent at an angle of ninety degrees 5 mm. from the center of the head. When the handle is rotated between the fingers on its long axis, the head will describe a circle of 1 cm., and will ap-

pear to be moving back and forth over a linear distance of 1 cm. In this way the amount of motion of the test object is accurately controlled.

J. Hewitt Judd.

Wood, E. H. **Study of transillumination of the eye.** Arch. of Ophth., 1939, v. 22, Oct., pp. 653-666.

This review traces the history of transillumination and transilluminators from Graefe to the present time, and gives a summary of the uses of transillumination in modern ophthalmic practice. (Bibliography.)

J. Hewitt Judd.

2

THERAPEUTICS AND OPERATIONS

Bader, Alfred. **Successes and failures of roentgen therapy.** Klin. M. f. Augenh., 1939, v. 103, Oct.-Nov., p. 503.

A case of suprasellar brain tumor and one of posttraumatic cyst of the iris were treated with roentgen rays and apparently were rapidly and definitely cured. After five and four years respectively relapses occurred which did not react to renewed radiation.

C. Zimmermann.

Glees, M. **The effects of a new imidazolin (Priscol-Ciba) upon the eye.** Graefe's Arch., 1939, v. 140, pt. 3, pp. 497-501.

The new aromatic imidazolin, Priscol, is a powerful vasodilator, acting upon the blood vessels of the conjunctiva and iris. The author has studied the physiologic effects of the drug after instillation into the conjunctival sac and after parenteral injection. Priscol is chemically similar to histamine and pilocarpine, but it dilates the pupil. It has a parasympathetico-paralytic effect upon isolated iris muscle, but does not influence intraocular pressure.

Charles A. Perera.

Jones, Cecil C. **The use of sulphanilamide in otolaryngology and ophthalmology.** Iowa State Med. Jour., 1939, v. 29, Jan., p. 6.

The history and chemical composition of the drug are given; the bacteria affected and the disease processes in which the drug is indicated are mentioned. Among the diseases in which it is considered a specific are streptococcic orbital cellulitis, gonorrheal ophthalmia, and gonococcic ulcers of the cornea. The method of administration is carefully considered and particular study is made of the toxic manifestations. The author concludes that sulphanilamide is a valuable adjunct to our present armamentarium, but its use must be limited to proved indications. Advice is given regarding contraindication to its use, toxic manifestations, and the essential need for routine blood counts during the administration of the drug.

F. M. Crage.

Mengel, W. G. **Determination of sulphanilamide in aqueous and vitreous after conjunctival and oral administration.** Arch. of Ophth., 1939, v. 22, Sept., pp. 406-409.

The investigations were made on blind eyes, one with absolute glaucoma and the others with phthisis bulbi. The amount of sulphanilamide absorbed into the aqueous after local instillation into the conjunctival sac was less than 0.1 mg. per 100 c.c. whereas sulphanilamide was found in the aqueous and vitreous in a concentration of 1.5 to 3.2 mg. per 100 c.c. after administration by mouth. Sulphanilamide was found in the vitreous in a concentration of 1.1 mg. per 100 c.c. 32 minutes after

administration by mouth. Because of its diffusion in the ocular fluids this drug is considered very valuable for treating infections in the eye.

J. Hewitt Judd.

Paton, R. T. **Subconjunctival injections of neoprontosil in the treatment of ocular infections.** Arch. of Ophth., 1939, v. 22, Sept., pp. 377-384.

Local administration of sulphanilamide apparently produces some effective mechanism. Neoprontosil, administered conjunctivally every second day for several doses of 0.3 to 0.5 c.c. of a 2.5-percent solution, was well tolerated in the presence of certain severe intraocular and extraocular infections such as iritis, scleritis, interstitial keratitis, trachoma, and corneal ulcers. If there had been no improvement after the third injection, little if any could be expected by subsequent injections. The results in some of the forty treated patients are tabulated. For ambulatory patients, in whom daily blood count and determination of hemoglobin and sulphanilamide concentration are impracticable, combined oral and subconjunctival injections lessen the danger of severe toxic effects. (Discussion.) J. Hewitt Judd.

Phillips, T. J. **The therapeutic use of short-wave diathermy in ophthalmology.** Trans. Ophth. Soc. United Kingdom, 1939, v. 59, pt. 1, p. 193.

The author advises the use of short-wave diathermy in conditions of deep inflammation. The instrument is portable, and the electrode is a flexible rubber-covered copper cable, easily adjusted to the patient's eye. There is practically no risk of burn, and the depth of heat may be controlled by varying the distance of the electrode from the patient. Beulah Cushman.

Stewart, Marshall. **A simple method of producing anesthesia during removal of transplants of mucous membrane.** Arch. of Ophth., 1939, v. 22, Oct., pp. 667-669.

Regional anesthesia whereby mucous-membrane grafts can be removed from the lip or cheek without pain, is produced by infiltration of procaine hydrochloride in the labial fold anterior to the incisor teeth, thus blocking the mental nerve. Mucous membrane of the cheek can also be blocked by anesthetizing the upper and lower nerve filaments within the mouth in the buccal sulci. J. Hewitt Judd.

Sukhov, S. **Dionin in diseases of the posterior ocular segment.** Viestnik Opht., 1939, v. 15, pt. 2, p. 18.

This is a clinical report on the use of subconjunctival injections of dionin in diseases of the posterior ocular segment. The results on thirty patients led the author to the conclusion that dionin should be among the agents used to absorb vitreous opacities and retinal hemorrhages. He prefers subconjunctival administration because it permits exact dosage. The dilatation of blood vessels and the accompanying lymphatic activity promote tissue nutrition and absorption of inflammatory exudates. Ray K. Daily.

Weve, H. **On diathermy in ophthalmic practice.** Trans. Ophth. Soc. United Kingdom, 1939, v. 59, pt. 1, p. 43.

After a historical survey of the use of the electric current and a description of the various types of apparatus, the applications of diathermy to such eye conditions as xanthelasma, tumor of the lids, intraocular inflammation, tuberculous lesion, angiomatosis retinae, intraocular tumor, and separation of

the retina are mentioned. (Illustrations.) Beulah Cushman.

3

PHYSIOLOGIC OPTICS, REFRACTION AND COLOR VISION

Adamson, J., Fincham, E. F. **The effect of lenses and convergence upon the state of accommodation of the eye.** Trans. Ophth. Soc. United Kingdom, 1939, v. 59, pt. 1, p. 163.

The authors report the results obtained after a series of readings in normal individuals between the ages of 18 and 46 years. They found that changes in binocular parallax with fixed-light vergence caused a change in accommodation less in amount than the change in convergence. Changes in incident-light vergence with fixed binocular parallax produced a preliminary stage in which there was no change in accommodation. Beulah Cushman.

Berens, Conrad. **A prism scale.** Amer. Jour. Ophth., 1940, v. 23, Jan., pp. 81-82.

Comberg, W. **Test types with reduced contrast between test objects and background as further means for determining vision.** Klin. M. f. Augenh., 1939, v. 103, Oct.-Nov., p. 466.

To avoid the extreme contrast between test objects and the white paper background, Comberg constructed charts, the background of which gave only 20 or 10-percent reflection as compared with the 75-percent reflection of the usual white background.

C. Zimmermann.

Cowen, J. P. **Cosmetic correction of facial asymmetry with prisms.** Amer. Jour. Ophth., 1940, v. 23, Jan., pp. 60-62.

Eggers, Harry. **Suggestions for a new design of stock contact lenses.** Arch. of Ophth., 1939, v. 22, Sept., pp. 403-405.

Based on examination of 200 casts, it was found that nearly all the eyes had a parabolic rather than a spherical profile, and that the profile was seldom symmetric. The author suggests the construction of a number of standard permanent steel models from which lenses can be molded. A table of 35 sizes of proposed standard curves in four profiles is given. It is also suggested that stock lenses be made available in two series in which the intervals between lenses are 2 mm. and 2.5 mm. respectively. J. Hewitt Judd.

Ferree, C. E., and Rand, G. **Attachment to the Ferree-Rand perimeter for determining light and color minima.** Arch. of Ophth., 1939, v. 22, Oct., pp. 636-652.

The authors review the three bases which have been used to measure light and color sensitivity: (1) light and color minima and light and color differences, (2) limits of sensitivity (the perimetric method), and (3) light minimum for the discrimination of detail. Because of the value of using minima and limits in clinical studies of the visual field, an attachment to the Ferree-Rand perimeter has been devised by means of which both the light and color minima can be determined easily and conveniently at any point in the field and the use of limits extended to include stimuli of different intensities in a continuously graded series. Some preliminary determinations of minima made with this attachment are given to show sensitivity gradients for light and color from center to periphery and variations in these gradients which may be caused by pathologic disturbances. A discussion is given of the value of the attachment as supplementary

equipment for the study of the visual field by the use of limits, especially in those cases where detection of slight changes, advances or recessions, is needed. It is also useful in studying the limits of the visual field under different intensities and illuminations and the effect of adaptations on these limits, in testing the peripheral vision of night flyers, and in cases in which an extension of the versatility of the perimetric method is wanted beyond that given by variation of size of stimulus and relation to background.

J. Hewitt Judd.

Goulden, Charles. **Equipment necessary for examining and correcting errors of refraction.** Trans. Ophth. Soc. United Kingdom, 1939, v. 59, pt. 1. p. 81.

Goulden discusses equipment for testing visual acuity for distance and near, illumination, test lenses and frames, source of light used in retinoscopy, and the value of testing the near point, muscle balance, and amplitude of convergence. Beulah Cushman.

Greeves, R. A. **Problems of refraction.** Trans. Ophth. Soc. United Kingdom, 1939, v. 59, pt. 1, p. 105.

Greeves discusses the fitting of lenses, with special emphasis upon the care necessary for the correction of amblyopia, monocular aphakia, and the phorias associated with myopia and hypermetropia. Beulah Cushman.

Haab, O. **Auto-observation of the eye. Entoptic phenomena by means of the slitlamp.** Klin. M. f. Augenh., 1939, v. 103, Oct.-Nov., p. 480.

By means of the slitlamp it is possible to observe some entoptic phenomena: (1) Entoptic perception of diffusely dispersed light from the point-illuminated cornea is seen as a simple dispersion without reflection. (2) Entoptic perception of light from the point-illuminated iris is seen as a diffuse dispersion with regular reflection from the anterior corneal surface. (3) A part of the focally illuminated lens is entoptically perceptible as a reflection from the anterior corneal surface. (4) Entoptic perception of the pupillary movements occurs. The optical conditions are discussed in detail.

C. Zimmermann.

Hartinger, H. **A new spectacle frame and test lenses.** Deutsche Ophth. Gesell., 1938; in Klin. M. f. Augenh., 1938, v. 101, July, p. 109.

The new Zeiss trial case contains punctal lenses with a working diameter of 15 mm. and an outer diameter of 37.5 mm. The lenses are light and thin. The red and black dioptric markings are readily distinguished. Spherical and cylindrical lenses, and prisms are recognized by special forms of the handle. The frame is light and simply constructed. Four lenses can be mounted in each cell. Percy Fridenberg.

Herzau. **A new Ames tilting plane.** Deutsche Ophth. Gesell., 1938; in Klin. M. f. Augenh., 1938, v. 101, July, p. 109.

Herzau's simplified demonstration model consists of a single plate which can be tilted at will, but is easily returned to the accurate horizontal position. Unilateral magnification, as by means of a lens, induces the subject to tilt the field forward on the side of the eye which has the larger image, in order to cause it to appear horizontal again. A difference of one percent in size of image corresponds to a four-degree inclination of the plane. This phenomenon is not altered by habituation but can be reduced or made to dis-

appear by bringing into play elements of perspective, as for instance placing a small, round, cardboard box in the tilting field. Percy Fridenberg.

Jablonski, Walter. **Investigation of the vision of ametropes.** Ophthalmologica, 1939, v. 98, Sept.-Oct., p. 56.

Myopes and hypermetropes without astigmatism were used for this investigation. In explaining psychologic-optical phenomena, it is important to distinguish distant stimulus from near stimulus. It was found that ametropic subjects who wear glasses constantly see objects more nearly correctly with their glasses than those who wear glasses intermittently. Those who wear glasses intermittently see better without glasses than do those habituated to glasses. The results are produced by factors other than so-called visual acuity. Great care must be exercised to ascertain to what extent the artificial conditions used in psychologic experiments correspond to the conditions of natural life. F. Herbert Haessler.

Krauss, Stephen. **The perception of illumination; a newly discovered function of the rods.** Acta Ophth. Orientalia, 1939, v. 1, April, p. 166.

Recent experiments with colors seen through artificially varied optical media demonstrated that the Purkinje phenomenon could be observed in daylight. Since alterations of intervening optical media are equivalent to changes of illumination in twilight, the author concludes that the rods must have a function in addition to that of twilight vision, namely perception of the optical medium or of illumination. Perception of illumination, the author argues, is an independent function carried on by the rods. R. Grunfeld.

Levy, A. H. **Methods of estimating refraction.** Trans. Ophth. Soc. United Kingdom, 1939, v. 59, pt. 1, p. 95.

The author discusses the possibilities of the ophthalmometer, the refractionometer, the optometer, and the duochrome test. Beulah Cushman.

Livingston, P. C. **Difficult refraction in relation to ocular muscle imbalances.** Trans. Ophth. Soc. United Kingdom, 1939, v. 59, pt. 1, p. 129.

The author states that probably the principal cause of visual hyperesthesia is ocular muscle imbalance, and that in dealing with these cases success can be attained only if the relative merits of refractive and orthoptic treatment are clearly understood. Beulah Cushman.

Lythgoe, R. J. **The mechanism of dark adaptation.** Brit. Jour. Ophth., 1940, v. 24, Jan., pp. 21-42.

The author states that no attempt is made to criticize the techniques of measurement, but rather to test the adequacy of theories and to explain results attained. In those countries experiencing the "blackout" periods due to war, the subject has assumed greater importance. Nervous influences may also modify dark adaptation. (Figures, references.) D. F. Harbridge.

McMullen, W. H. **Some points in anisometropia.** Trans. Ophth. Soc. United Kingdom, 1939, v. 59, pt. 1, p. 119.

The author states that the prescribing of glasses that will make a patient comfortable is an art which cannot be based solely on scientific measurements. In addition to the physical condition of the eyes, the patient's mentality, symptoms, and the nature of his work must be considered.

Beulah Cushman.

Majewski, K. W. **Exophoria in presbyopes.** Ann. d'Ocul, 1939, v. 176, Oct., pp. 744-747.

Considering the normal accommodation-convergence relationship, a certain amount of exophoria should be considered physiologic in otherwise normal presbyopes.

John M. McLean.

Mendoza, R. **Errors in physiologic optics.** Rev. Oto-Neuro-Oft., 1939, v. 14, Jan., p. 31.

The author is not in accord with some explanations commonly given in modern textbooks with regard to the behavior of images, and he offers alternate explanations. Edward P. Burch.

Ogle, K. N. **Induced size effect. 3. A study of the phenomenon as influenced by horizontal disparity of the fusion contours.** Arch. of Ophth., 1939, v. 22, Oct., pp. 613-635.

For this study it was necessary to devise a combination haploscope and test-plane arrangement wherein the reflected images of simple patterns could be adjusted for given horizontal disparities relative to the center of the plane. In the experiments taken with the inclined test plane, the inclination introduced horizontal disparity of the images automatically, whereas with a combination of haploscope and rotatable test plane, specific horizontal disparities of the central fusion patterns on the haploscope targets were introduced deliberately. It was found that the phenomenon of induced size effect decreased rapidly when horizontal disparities were introduced between the images of the fusion contours. This decrease followed the same general course whether these disparities were introduced simply, as in the haploscope experiments, or complexly, as in inclined test surface experiments. The greatest response of the eyes to the induced effect occurred when the retinal images of the fusion contours fell nearest to the corresponding retinal points. The findings suggested that the attraction or the influence between horizontally disparate images decreased fractionally with the increase of horizontal disparity. J. Hewitt Judd.

Pascal, J. I. **Form and power of contact lenses.** Arch. of Ophth., 1939, v. 22, Sept., pp. 399-402.

Contact lenses are always deep meniscus and plano in effect whereas ordinary test lenses are either double-curved or plano-curved and have a definite plus or minus power. The author describes and shows by means of a graph how it is possible to change a meniscus lens with fixed radii and concave both in form and effect to a lens which is convex in form and effect, by merely changing the thickness. Between these two extremes is a lens which is plano in form and concave in effect, one which is convex in form and concave in effect, and one which is convex in form and plano in effect.

J. Hewitt Judd.

Pascal, J. I. **A memory scheme for the cardinal points.** Arch. of Ophth., 1939, v. 22, Sept., pp. 448-449.

As an aid to memorizing the six cardinal points a benzene ring is used. The two principal focal points are placed one at the top and one at the bottom of the ring. The two principal points are placed at one side and the two nodal points are placed at the other side of the ring. The lines which are parallel to one another then represent equal distances, and the three equations become apparent. J. Hewitt Judd.

Pascal, J. I. **A simple method of calculating lens power.** Amer. Jour. Ophth., 1940, v. 23, Feb., pp. 187-190.

Problems of refraction, a symposium on. See Goulden, Greeves, Levy, Livingston, McMullen, and Terrien and Onfray.

Taliercio, Aurelio. **The external accommodative effect in corrected ametropes.** Ann. di Ottal., 1939, v. 67, Sept., p. 668.

In an emmetrope and a corrected ametrope having equal accommodative power, the near points occupy different positions as a result of the influence of the lens correction of the ametrope. This effect of correcting lenses on the accommodation has been termed "external accommodation." The author discusses the optical principles involved and applies them in a series of clinical observations. (Bibliography.)

Park Lewis.

Terrien, F., and Onfray, L. **Use and value of the diploscope in routine refraction.** Trans. Ophth. Soc. United Kingdom, 1939, v. 59, pt. 1, p. 125. (See Amer. Jour. Ophth., 1939, v. 22, Dec., p. 1419.)

Tschermak-Seysenegg, A. **Contribution to the functional organization of the retina.** Graefe's Arch., 1939, v. 140, pt. 3, pp. 445-455.

The author explains the functional organization of the retina, and compares the system of hyperboles which he found with that formulated by Helmholtz. Charles A. Perera.

Weinman, E. B., and Fralick, F. B. **Comparative study of benzedrine, paredrine, and cocaine with homatropine as cycloplegics.** Amer. Jour. Ophth., 1940, v. 23, Feb., pp. 172-178.

Williamson-Noble, F. A., Dallos, J., and Mann, I. **Sphero-cylindrical contact lenses—a preliminary note.** Brit. Jour. Ophth., 1940, v. 24, Jan., pp. 43-46.

As a result of recent improvements in technique, cylindrical correction is now possible in contact lenses. Both the theoretical and practical sides of the question are discussed. (Case reports, tables.) D. F. Harbridge.

Wright, W. D. **Response of the eye to light in relation to the measurement of subjective brightness and contrast.** Brit. Jour. Ophth., 1940, v. 24, Jan., pp. 1-20.

The subject is delineated, in the technical form necessary, under the three following sections: response of the eye to light, under which objective and subjective data are given; measurement of subjective brightness, under which the maintenance of a standard state of adaptation and a method for comparing apparent brightness are presented; and specification of contrast, under which a scale and measurement for contrast are outlined. (Figures, references.) D. F. Harbridge.

Yanes, T. R. **Hypovitaminosis A, its clinical demonstration.** Arch. Soc. Estudios Clin. Habana, 1939, June 8, pp. 1-16.

In a paper dealing with the relationship of retinal adaptation to deficiency in vitamin A, the author explains principles and instrumentarium, and gives rules for interpreting the biophotometric curves. While such tests are still in an experimental stage, they may prove valuable to the oculist in study of disturbances of twilight adaptation of the retina. W. H. Crisp.

4

OCULAR MOVEMENTS

Borsotti, Ippolito. **Considerations of a case of third-nerve paralysis with disturbances of deglutition and phonation.** Riv. Oto-Neuro-Oft., 1939, v. 16, Jan.-Feb., pp. 15-33.

During the course of a month a 44-year-old man developed paralysis of the left levator and internal rectus, and paresis of the superior rectus muscle, beside phonetic and deglutition difficulties. Specific treatment with bismuth and iodide effected a rapid improvement and after two years only a slight insufficiency of the internal rectus persisted. The author discusses disseminated sclerosis, cerebral lues, and an aspecific or toxic infectious neuritis as possible etiologic factors in this case. (Bibliography, 5 figures.)

M. Lombardo.

Heinsius. **Apparatus for measurement of cyclophoria.** Deutsche Ophth. Gesell., 1938; in Klin. M. f. Augenh., 1938, v. 101, July, p. 108.

Many cases of asthenopia which are not relieved by the most careful correction of ametropia are due to latent deviations from the normal rest-position of the globe. There is a simple and accurate apparatus for quickly determining horizontal and vertical phorias but none for determining cyclophoria, although the latter is comparatively frequent. In a series of 311 normal eyes in young subjects with not more than 1.5 D. of hyperpetropia, cyclophoria was found in 194 (65.6 percent). A modification of the Maddox-Stock phorometer is used in which the rectilinear component of the intentionally elicited combined image can be rotated and the angular degree read off on a scale. Any refractive error must be corrected as a preliminary, and care must be taken to have the patient's head held steady. The apparatus also measures the usual phorias and degrees of paralytic squint.

Percy Fridenberg.

Ivanov, A. I. **Technical modification of the strabismus operation utilizing aural cartilage.** Viestnik Opht., 1939, v. 15, pt. 2, p. 43.

The author uses two strabismus hooks instead of the double de Wecker hook in performing Dudinov's operation for strabismus. (Illustrations.)

Ray K. Daily.

Jemmi, Carlo. **An interesting case of peritubaric lymphosarcoma with intracranial diffusion.** Riv. Oto-Neuro-Oft., 1938, v. 16, July-Aug., pp. 336-348.

In a man 35 years of age, a lymphosarcoma which had its origin in the right pharyngeal region was characterized by rapid intracranial diffusion and by marked cervical metastatic adenopathy. The third and sixth nerves were affected, together with six other cranial nerves. A histologic description of the neoplasm is given. (Bibliography, 5 figures.)

M. Lombardo.

Livingston, P. C. **Difficult refraction in relation to ocular muscle imbalances.** Trans. Ophth. Soc. United Kingdom, 1939, v. 59, pt. 1, p. 129. (See Section 3, Physiologic optics, refraction, and color vision.)

Nugent, O. B. **Functional training, an aid in the surgical correction of strabismus.** Amer. Jour. Ophth., 1940, v. 23, Jan., pp. 68-72.

Odeneal, T. H. **The significance of the orbit level in muscle balance.** Amer. Jour. Ophth., 1940, v. 23, Feb., pp. 190-191.

Ohm, J. **Nystagmus.** 46th and 47th communications. Graefe's Arch., 1939, v. 140, pt. 2, pp. 379-389; and pp. 527-530.

The former paper deals with miners' nystagmus. The latter contains a report of a case in an albinotic patient with amblyopia. Charles A. Perera.

Rubino, A., and Quarti, M. **Ophthalmoplegic syndrome from aneurysm of the internal carotid artery in the cavernous sinus.** Riv. Oto-Neuro-Oft., 1939, v. 16, May-June, pp. 214-223.

Two months after a contusion on the head, a 42-year-old man developed a severe left hemicrania lasting about a month, and later, a nonpulsating exophthalmos with paresis of the third and fourth nerves. In addition he complained of a disturbing noise in the left ear. Transpalpebral auscultation revealed a systolic bruit which ceased on compressing the left carotid artery. Arteriographs showed an aneurysmal dilatation of the internal carotid at the level of the cavernous sinus. Ligation of the common carotid resulted in cessation of all symptoms. The authors discuss the clinical significance of the painful symptomatology and the diagnostic importance of the ophthalmoplegia. As to etiology they presuppose a peculiar congenital weakness of the walls of the bloodvessels. (6 figures.) M. Lombardo.

Trovati, Emma. **Increase in the area of the indirect visual field in one eye in relation to the binocular visual field in orthophoria and in concomitant external and internal strabismus.** Ann. di Ottal., 1939, v. 67, Sept., p. 681.

The author in these studies has endeavored to demonstrate that the visual field of one eye of a normal individual may be found to be of a lesser area than that of the fellow eye, and that this limitation will be found in the temporal half of the visual field and more frequently in the left eye. In concomitant internal strabismus, while the visual field of the fixing eye will be normal, the strabismic eye will consistently show a restriction of the nasal periphery. In concomitant external strabismus the visual fields of both eyes, and especially the left, will show concentric restrictions. Of the binocular visual fields in concomitant internal strabismus, the central area common to both eyes will be found normal but there will be temporal monocular limitation on the side of the strabismic eye. In concomitant external strabismus there will be a slight limitation of the central field common to both eyes, with some restriction of the temporal field of the strabismic eye. The present study with the strabismic as the fixing eye warrants the conclusion that this eye has a restricted function in the limits of the binocular field especially in internal concomitant strabismus. (Bibliography.) Park Lewis.

5

CONJUNCTIVA

Barbour, F. A., and Towsley, H. A. **Experience with sulphanilamide in treatment of gonorrheal ophthalmia.** Arch. of Ophth., 1939, v. 22, Oct., pp. 581-589.

The authors compare the results in 15 cases of gonorrheal ophthalmia in which sulphanilamide was employed with the results in 15 consecutive cases prior to the use of sulphanilamide. The results in 55 cases reported in the literature are also summarized and compared. It was found that the incidence of corneal complications was diminished, that hospitalization was reduced

to an average of 8.3 days, that under careful supervision the drug could be given with a minimum of complications, and that newborn infants tolerated and required larger doses of the drug than adults to raise the blood concentration to the expected level.

J. Hewitt Judd.

Braley, A. E. **Relation between the virus of trachoma and the virus of inclusion blennorrhea.** Arch. of Ophth., 1939, v. 22, Sept., pp. 393-398.

The virus of trachoma, like that of inclusion blennorrhea, produces in baboons a cervicitis which when transferred to the conjunctiva produces follicular conjunctivitis. One of the four baboons used for the experiment died three days after inoculation, but the three remaining were examined at intervals, and at no time were inclusion bodies present in the epithelial smears. The immediate response of the virus appeared to be the production of initial bodies. The epithelium becomes hyperplastic and hypertrophic before any evidence of inflammation is present. No conjunctival immunity is conferred by infection of the cervix with the virus, nor does the trachoma virus appear to be attenuated after growth in the cervix. The conjunctival epithelium and the transitional epithelium of the cervix appear to be the only tissues susceptible to infection with the virus of trachoma. Since the virus of inclusion blennorrhea and of trachoma produce similar pathologic conditions, a similarity in their nature appears probable.

J. Hewitt Judd.

Cordes, F. C., and Hogan, M. J. **Ocular ichthyosis.** Arch. of Ophth., 1939, v. 22, Oct., pp. 590-594.

A case of generalized ichthyosis of the skin in a woman aged 24 years is reported. There was bilateral involvement of the conjunctiva with involvement of the right cornea. The conjunctiva showed a thickening and marked redness without papillary or follicular formation. A pannus and infiltration extending from the lower limbic region into the center of the right cornea were probably secondary to the conjunctival lesion. Ichthyosis of the skin may involve the ocular tissues as asymptomatic dystrophy of the corneal cells and nerves; in an inflammatory form, showing smooth or papillary hyperplasia of the conjunctiva, with involvement of the cornea due to mechanical irritation; and as a primary keratosis of the cornea. Treatment is symptomatic.

J. Hewitt Judd.

Derkač, V. **Experiments with the vaccine of proteus X19 in trachoma.** Graefe's Arch., 1939, v. 140, pt. 3, pp. 502-506.

The author discusses the Weil-Félix reaction in rickettsial diseases and reviews the evidence that trachoma belongs to this group. He found a positive Weil-Félix reaction in many patients suffering from trachoma. These findings were confirmed by Pŏstić and Poleff. Nicolle was unable to immunize patients against typhus by the use of proteus X19 vaccine. The author records his results after injecting this vaccine intracutaneously and subcutaneously in 200 patients of whom 173 were trachomatous. He found that the reaction to the injections was the same in trachomatous and in nontrachomatous patients, and that the intensity of the reaction had no diagnostic significance. The use of proteus vaccine in the treatment of trachoma showed its benefits to correspond to foreign-protein therapy, and without specific effect.

Charles A. Perera.

Georgiadès. **Resorption granulomas of the lids and conjunctiva.** Ann. d'Ocul., 1939, v. 176, Sept., pp. 675-701. (See Section 14, Eyelids and lacrimal apparatus.)

Jebejian, Robert. **Observations on the effect of sulphanilamide taken internally for trachoma.** Ann. d'Ocul., 1939, v. 176, Sept., pp. 671-575.

In trachoma cases sulphanilamide in comparatively small doses has immediate beneficial effect on symptoms of irritation, lacrimation, photophobia, and blepharospasm. These symptoms disappear in large measure after about three days of treatment. Pannus is without doubt favorably influenced and begins to regress in a steady fashion with resultant improvement of vision. Keratitis and corneal infiltrations are also favorably affected. Conjunctival vascularization decreases appreciably, but hypertrophic lesions and granulations are not noticeably affected. Maximum therapeutic effect is attained in a few days and continuation of treatment after this period does not appear effective. Sulphanilamide has an active inhibition on the trachoma process and prepares the field for more effective treatment. Sulphanilamide is not a cure but is a valuable adjunct and fills an important place together with other standard treatments. In cases which are resistant to classical treatment a short course of sulphanilamide is particularly indicated. (8 case reports.)

John M. McLean.

Julianelle, L. A., and Smith, J. E. **Studies on the infectivity of trachoma. 9. Immunological aspects of the rickettsial concept.** Amer. Jour. Ophth., 1940, v. 23, Jan., pp. 62-67.

Katznelson, A. B., Kachan, Z. Z., and Tufa, H. Z. **The pathogenesis of phlyctenules.** Viestnik Opht., 1939, v. 15, pt. 2, p. 3.

The objective of this investigation was experimental demonstration of the significance of carbohydrate metabolism in tuberculo-allergic ocular inflammations. Rabbits sensitized to tuberculin and subsequently desensitized were given 20-percent sodium lactate, 2 grams per kg. of weight; and a solution of tuberculin was instilled into the conjunctival sac 15 to 20 minutes later. In all six rabbits thus treated there was a reactivation of sensitivity to the local introduction of tuberculin. Rabbits sensitized to tuberculin, and not subsequently desensitized, developed a conjunctival inflammation following the consumption of the sodium lactate, without the introduction of the allergen into the conjunctival sac. Normal rabbits given the lactate responded to conjunctival instillations of tuberculin with a brief catarrhal inflammation. In all animals consuming the sodium lactate the blood showed a pronounced rise in lactic acid; the sugar content did not show any significant variations. These studies indicate that changes in carbohydrate metabolism have a definite role in the pathogenesis of tuberculo-allergic diseases, and that the rise of lactic acid in the blood is the cause and not the result of these conditions.

Ray K. Daily.

Kirk, R., McKelvie, A. R., and Drysdale, A. D. **The Weil-Félix reaction in trachoma.** Jour. of Hygiene, 1939, v. 39, Sept., pp. 553-557.

The impression that trachoma is due to a rickettsia organism closely related to typhus has led to several attempts to demonstrate the Weil-Félix reaction in trachomatous patients. The published results have been conflicting. The authors examined the sera of 200

trachomatous patients in the Sudan and used as controls 22 nontrachomatous individuals and 1,000 others selected without reference to trachoma. The Weil-Félix reaction was negative in all cases except in very low dilutions and there was no significant difference in reaction between trachomatous and nontrachomatous individuals.

John C. Long.

Lipovskaja, A. I. **Gummatous involvement of the conjunctiva and lids.** Viestnik Opht., 1939, v. 15, pt. 2, p. 88.

A report of three cases. One, which took the form of a conjunctival ulcer, had at first been treated for tuberculosis; but absence of glandular enlargement, with positive serology and therapeutic response to antiluetic therapy, justified a diagnosis of syphilis. The second case was one of ulcer of the lid, treated first as a stye complicating ulcerative blepharitis; serologic tests indicated the nature of the lesion and it healed under antiluetic therapy. The third case was one of gumma of the upper lid, with bilateral interstitial keratitis. Ray K. Daily.

Mariani, G. **Technical details for studying the rickettsioses.** Minerva Med., 1939, v. 2, Nov., pp. 433-447.

The author is director of the Laboratory for the Prophylaxis and Study of the Rickettsioses, recently established at Addis-Ababa, Italian Africa. The details, which are described elaborately, and with an excellent series of illustrations, include the breeding of the necessary lice, biologic tests for disintegration of species, methods of staining the rickettsias, emulsions used for vaccination, and cultures in vitro. (Bibliography.) W. H. Crisp.

Nicholls, J. V. V. **Epithelial plaques of the conjunctiva and the cornea.** Arch. of Ophth., 1939, v. 22, Sept., pp. 370-376.

The literature is reviewed and a classification presented consisting of three types of epithelial plaques: (1) those in patients suffering from xerosis, (2) those of congenital origin, and (3) those resulting from chronic irritative conditions of the conjunctival sac. The third type is subdivided into those showing cornification and epithelial hyperplasia of moderate degree and those with marked changes. A case of epithelial-plaque formation in a boy 14 years of age is reported. The growth was excised and studied histologically. It showed an intense epithelial hyperplasia bordering on neoplasia, and essentially similar to that of leukoplakia. It appeared to be a precancerous condition. J. Hewitt Judd.

Rameev, P. C. **Therapy-resistant types of trachoma.** Viestnik Opht., 1939, v. 15, pt. 2, p. 34.

For cases resistant to all therapeutic and surgical procedures, Rameev recommends a complete pause in local medication combined with a general rest and change of climate. He believes that the ocular tissues reach a saturation stage and further treatment leads only to tissue irritation and destruction. Ray K. Daily.

Rötth, A. de. **The problem of the etiology of trachoma. 1. Rickettsia.** Arch. of Ophth., 1939, v. 22, Oct., pp. 533-539.

The literature is reviewed and the results of experiments are reported. The author was unsuccessful in repeating the Cuénod and Nataf experiments of infecting monkeys with lice which had been infected with trachomatous material by the anal route. Thus by other means he arrived at the same

conclusion reached by Thygeson that the so-called rickettsia are not parasitic but in all probability cell granules and cytoplasmic debris. J. Hewitt Judd.

Streiff, E. B. **Therapeutic consideration of the action of bee venom in trachoma.** Ann. di Ottal., 1939, v. 67, Oct., p. 776.

The author bases his therapeutic experiments on the following premises: that the retino-endothelial system is involved in the majority of cases of trachoma, as shown by the use of colored substance; that blood-chemistry studies have shown that a hypercholesteremia exists in trachoma, and that the secretion of cholesterin is dependent on the activity of the retino-endothelial membrane; that cholesterin is antibacterial, antitoxic, antihemolytic, and anti-infective; that trachoma and rheumatism are both allergic manifestations; and that, as an antirheumatic, bee venom should have therapeutic value in trachoma. As none of these postulates has been scientifically proved, the value of the use of the venom is not convincing, especially as the substance was locally applied to the upper and lower tarsal conjunctiva and the effects were those that would follow the use of any local irritant. Moreover, as trachoma is not common in Geneva, the numbers treated were not sufficient to constitute a positive demonstration. (Bibliography.)

Park Lewis.

6

CORNEA AND SCLERA

Abdulaev, G. G. **Tissue homotransplantation in trachoma, and its complication.** Viestnik Opht., 1939, v. 15, pt. 2, p. 52.

The author uses preserved cadaver cornea, conjunctiva, and sclera, and cadaver mucous membrane from the lip in trachomatous corneal opacities and pannus. In 15 cases of corneal transplantation observed over a period of from two to three months the visual results were satisfactory.

Ray K. Daily.

Comberg. **Keratitis superficialis vorticosa.** Deutsche Ophth. Gesell., 1938; in Klin. M. f. Augenh., 1938, v. 101, July, p. 110.

Demonstration by pictures of four cases, evidently herpetic, with a definite whirlpool figure having its center near the middle of the cornea. In two cases, the lesion was of an infectious nature in connection with a common cold, and quickly subsided. In two chronic cases, the vortex figure was more marked. In one, corneal sensitivity was lowered and there was superficial punctate keratitis. There was a history of herpes zoster on the same side of the head. Percy Fridenberg.

Cordes, F. C., and Hogan, M. J. **Ocular ichthyosis.** Arch. of Ophth., 1939, v. 22, Oct., pp. 590-594. (See Section 5, Conjunctiva.)

Cornet, Emmanuel. **Epithelialized descemetocele.** Ann. d'Ocul., 1939, v. 176, Nov., pp. 823-824.

Epithelialized descemetocele is a rare but not pathognomonic condition, although it is found more frequently in the course of trachoma. It consists of complete ulceration of the corneal tissue down to Descemet's membrane. The membrane, however, does not rupture, but is slowly covered over by a layer of corneal epithelium, leaving a small area in the cornea consisting of three layers: epithelium, Descemet's membrane, and endothelium without any intervening stroma. There is little

danger of rupture unless this area is exposed to sudden severe strain.

John M. McLean.

Cuesta Yañez, Carlos. **Treatment of pannus and trachomatous keratitis with sulphanilamide.** La Semana Med., 1939, v. 46, Nov. 2, pp. 1048-1050.

Six clinical cases are cited. Intramuscular doses of sulpharsenol were given at the same time. The pannus, of variable amount, is said to have cleared up in respective periods of from one to two weeks. The sulphanilamide was used "intensively" (dose not clearly stated.) W. H. Crisp.

Feldman, A. M., and Medvedev, H. I. **Ocular inflammations in combination with Boeck's sarcoid.** Viestnik Opht., 1939, v. 15, pt. 2, p. 49.

Report of a case of recurrent keratoscleritis of the left eye in a woman 32 years old, who for two years had been affected by Boeck's sarcoid of the skin. In the course of the ocular disease there developed anterior synechia, opacity of the lens capsule and vitreous opacities. The ocular recurrences were coincident with recurrence of the skin lesions.

Ray K. Daily.

Gallardo, E., and Thompson, R. **The effect of the intravenous injection of nonspecific proteins upon experimental corneal ulcers.** Amer. Jour. Ophth., 1940, v. 23, Feb., pp. 127-137.

Gallardo, E., Pfeiffer, R., and Thompson, R. **Grenz-ray treatment of experimental infection of the cornea.** Amer. Jour. Ophth., 1940, v. 23, Jan., pp. 41-47.

Kaelin-Sulzer, Marguerite. **Two brothers with keratoconus.** Klin. M. f. Augenh., 1939, v. 103, Oct.-Nov., p. 513.

In two brothers the keratoconus showed corresponding form and equal extension of opacity.

C. Zimmermann.

Kiehle, F. A., and Darnell, C. A. **Reduplication of Descemet's membrane.** Arch. of Ophth., 1939, v. 22, Oct., pp. 672-674.

A girl aged 13 years presented a uniform band of almost transparent membrane extending obliquely across the pupil within the anterior chamber. This had resulted from a birth injury. Its appearance and the probable sequence of steps in its formation are shown by drawings. The causation lay in the splitting of Descemet's membrane by a forceps compressing the globe against the bony wall. J. Hewitt Judd.

Klainguti, R. **Primary ribbon-shaped degeneration of the cornea.** Klin. M. f. Augenh., 1939, v. 103, Oct.-Nov., p. 478.

For fifteen years the author observed a man of 53 years with primary ribbon-shaped opacities of the cornea of each eye. In previously reported cases the pathologic basis was found to be hyaline degeneration with lime deposits in Bowman's membrane.

C. Zimmermann.

Kyrieleis, Werner. **Circumscribed scleromalacia in old age.** Klin. M. f. Augenh., 1939, v. 103, Oct.-Nov., p. 441.

In three patients of advanced age thinning of the upper strata of the sclera was found. Anatomically one of these eyes showed disappearance of the superficial fibers and a massive calcification of the sclera. The general condition of the patients would indicate that chronic rheumatic diseases favor the changes. Senile alterations of metabolism and of the blood vessels also seem to be important. C. Zimmermann.

Levy-Wolff, Lizzie. **Prevention of keratitis punctata.** Ophthalmologica, 1939, v. 98, Sept.-Oct., p. 51.

To avoid keratitis punctata as a complication of conjunctivitis, and particularly of conjunctivitis caused by the Koch bacillus, it is only necessary to eliminate silver therapy. This is definitely shown by statistics for a year. The author points out that all metals with dehydrating or oxidizing tendency must be avoided in order to retain the reduction potential necessary for the preservation of corneal transparency. F. Herbert Haessler.

Makarov, H. H. **A rare case of neuroparalytic keratitis, following a phlegmon of the neck.** Viestnik Opht., 1939, v. 15, pt. 2, p. 97.

A woman 22 years old had a phlegmon of the left side of the neck and subsequently developed anesthesia of the left side of the face and neuroparalytic keratitis. The toxins in the neck had probably spread along the carotid artery into the gasserian ganglion.

Ray K. Daily.

Meesmann, A., and Wilke, F. **Clinical and anatomic investigations of a previously unknown dominantly inherited dystrophy of the corneal epithelium.** Klin. M. f. Augenh., 1939, v. 103, Oct.-Nov., p. 361.

The clinical picture of a previously unknown epithelial dystrophy of the cornea is exactly defined and the differential diagnosis from other types discussed. The slitlamp shows punctiform and vesicular opacities of the epithelium with slight roughness of the surface. The condition commences in earliest childhood and progresses with age; there is pain but not much impairment of vision. The deeper strata are not involved and no relation to the corneal nerves can be proven. Yellow discoloration of the scars is early observed. Hereditary transmission occurred in 34 patients of two genealogic trees and was dominant.

C. Zimmermann.

Muir, E. B. **Salzmann"s nodular corneal dystrophy.** Amer. Jour. Ophth., 1940, v. 23, Feb., pp. 138-144.

Nicholls, J. V. V. **Epithelial plaques of the conjunctiva and the cornea.** Arch. of Ophth., 1939, v. 22, Sept., pp. 370-376. (See Section 5, Conjunctiva.)

Panchenko, V. D. **Bilateral familial megalocornea.** Viestnik Opht., 1939, v. 15, pt. 2, p. 91.

This condition was found in a 45-year-old man who came for a cataract extraction. He stated that his mother and maternal grandfather also had large eyes. Ray K. Daily.

Piccollo, P. **Dystrophic degenerative adiposis of the cornea.** Ann. di Ottal., 1939, v. 67, Oct., p. 721.

For many years degenerative corneal changes with whitish or yellowish-white deposits in the form of spots in the superficial membranes have been noted and have been recognized as amyloid or fatty deposits from the organic fluids. These have been more frequently observed in absolute glaucomatous eyes and others with marked destructive changes. Primary degenerative deposits have, however, been exceedingly rare. Such cases as have been described are reviewed by the author and the histopathologic picture given. He adds two new cases, one of a woman of twenty and the other of a man of 62 years. In the light of our present knowledge these cases are fully discussed and illustrated by plates. (Bibliography, 1 colored plate, 4 figures.)

Park Lewis.

Schnyder, W. F. **Disciform deposits of crystals in the corneal center as a hereditary affection.** Klin M. f. Augenh., 1939, v. 103, Oct.-Nov., p. 494.

Schnyder describes four cases and gives the history of two deceased members of the same family, in all of whom bilateral crystalline opacities had existed since early childhood in the center of the cornea, without inflammation or other signs of irritation. The crystal impregnations were mostly in the anterior third of the parenchyma, immediately below Bowman's membrane. In all the cases a white limbal zone of Vogt's type 1 was present at the nasal and temporal limbus within the interpalpebral fissure. This previously unknown type of corneal degeneration is inherited dominantly.

C. Zimmermann.

Vianna, A. M. **A case of ring abscess of the cornea.** Ann. d'Ocul., 1939, v. 176, Nov., pp. 824-827.

A case report of cataract extraction on an only eye with severe infection discovered on the first postoperative day. A ring abscess of the cornea developed and the eye was finally exenterated because of panophthalmitis. Cultures were not made, but a single smear showed extracellular diplococci.

John M. McLean.

7

UVEAL TRACT, SYMPATHETIC DISEASE, AND AQUEOUS HUMOR

Crecchio, A. de, and Brignola, D. **The alkaline reserve in the blood and the pH of the aqueous in experimental acidosis.** Ann. di Ottal., 1939, v. 67, Oct., p. 781.

The author made studies of the alkaline reserve of the blood and the pH of the aqueous in provocative experimental acidosis in the rabbit. The acidosis was followed by oral administration of dilute hydrochloride acid, and the degree paralleled the diminution in the pH of the aqueous. (Bibliography.)

Park Lewis.

Goldfeder, A. E., and Byshmich, G. G. **Rare forms of spontaneous cyst of the iris.** Viestnik Opht., 1939, v. 15, pt. 2, p. 21.

After referring the reader to Granström's article on the subject (Amer. Jour. Ophth., 1930, v. 13, p. 654) for the cases reported prior to 1930, the authors review the literature since 1930 and report two cases of their own. In a 30-year-old woman, the cyst was on the posterior surface of the iris. The diagnosis between sarcoma and cyst was made with the slitlamp, which demonstrated the liquid contents of the cyst. Outside of producing an irregularity of the pupil it caused no difficulty. After an 18-month period of observation it disappeared spontaneously. The patient was engaged in intensive study, and the authors assume that the contractile movements of the intraocular muscles caused a rupture of the cyst with an evacuation of its contents. The second patient came to the hospital with an ulcer of the left cornea, and routine examination revealed a brown cyst attached to the lens in the lower medial quadrant of the anterior chamber. There was evidence of an old iridocyclitis and the authors believe that this cyst developed in a posterior synechia and subsequently was torn free from the iris. (Illustrations.)

Ray K. Daily.

Rones, Benjamin. **Essential atrophy of the iris.** Amer. Jour. Ophth., 1940, v. 23, Feb., pp. 163-171.

8

GLAUCOMA AND OCULAR TENSION

Evans, P. J. **The underlying causes of glaucoma.** Brit. Jour. Ophth., 1939, v. 23, Dec., pp. 745-783.

The author states that, although the subject has held the attention of ophthalmologists for many generations, only during the past fifty years has any substantial progress been made in elucidating the pathology of glaucoma. The author discusses the problem under the following headings: historical survey (from earliest records); recent theories (covering especially the past twenty years); the underlying causes of glaucoma; glaucoma in association with new growths; the vascular basis of primary glaucoma; chronic glaucoma; the course of future research; and numerous other pertinent topics. (Figures, bibliography.)

D. F. Harbridge.

Juler, Frank. **Hypotony after sclerocorneal trephining.** Trans. Ophth. Soc. United Kingdom, 1939, v. 59, pt. 1, p. 253.

The author discusses the literature and the three conditions associated with hypotony: detachment of choroid, pigmented exudation into the pupil, and late development of cataract. He describes his conjunctivoplasty, which he has used successfully in the restriction of hypotony in persistent cases.

Beulah Cushman.

Larsson, Sven. **Ophthalmoscopy through the hazy cornea in glaucoma.** Acta Ophth., 1939, v. 17, pt. 3, p. 297.

The author clarifies a central corneal segment by pressure with a small glass rod for from one half to one minute. This procedure clears the segment of edema sufficiently to permit ophthalmoscopy.

Ray K. Daily.

Lauber, H. **Combined operations for glaucoma.** Trans. Ophth. Soc. United Kingdom, 1939, v. 59, pt. 1, p. 267.

Lauder discusses the combination of cyclodialysis and iridectomy or trephining in glaucoma patients whose tension is not controlled by a single operative procedure. In cases of so-called posterior glaucoma, where the lens and iris are pushed forward, he has trephined the sclera posteriorly and aspirated the vitreous, and then performed iridectomy with satisfactory results.

Beulah Cushman.

Linksz, A., and Rasko, J. **Relationship between general and retinal arterial pressures and intraocular tension.** Ann. d'Ocul., 1939, v. 176, Oct., pp. 747-751.

The authors find Kukan's ophthalmodynamometer more satisfactory than Bailliart's or its various modifications. The results verify statements by Bailliart, Streiff, and others that the retinal blood pressure is one half the systemic pressure in normal individuals. They confirm the findings of Wesseley and Kukan that the intraocular tension is generally independent of rise or fall in systemic blood pressure. The diastolic retinal blood pressure seems to vary in a compensatory fashion with the intraocular pressure. There are a few exceptions to all of these findings which may be due to errors in measurement, emotional disturbance, unequal systemic blood pressure on the two sides of the body, or abnormalities in the autonomic regulation of the vascular system. This last possibility may give some lead in the study of migraine and neuralgia.

John M. McLean.

Lloyd, J. P. F. **The treatment of increased ocular tension with diathermy.** Trans. Ophth. Soc. United Kingdom, 1939, v. 59, pt. 1, p. 181.

The author used long-wave high-frequency diathermy for reduction of intraocular tension in 31 cases. In 8 patients the tension came down rapidly and remained so, in 17 the tension was reduced to the extent that an immediate operation was deferred and pain was reduced or abolished, in 4 the tension was reduced temporarily, and in 3 the tension either was not reduced or became worse.

Beulah Cushman.

Thomsen, Hugo. **The results of 244 glaucomatous eyes treated by Elliot trephining.** Acta Ophth., 1939, v. 17, pt. 3, p. 301.

In 87 of the 244 eyes, Bentzen's modification of the operation was used: a conjunctival flap was dissected up from the limbus and after trephining and iridectomy it was brought down over the cornea and sutured like a Kuhnt flap. There was no difference in result between the original and the modified techniques. Tension was reduced in 75 percent of all the cases, and in 91 percent of the glaucoma simplex cases. There were four cases of late infection.

Ray K. Daily.

9

CRYSTALLINE LENS

Bertoldi, Maria. **The elimination of ascorbic acid in the urine of cataract patients.** Rassegna Ital. d'Ottal., 1939, v. 8, Sept.-Oct., p. 572.

Bertoldi administered 300 mg. of ascorbic acid parenterally in 25 cataract cases, ranging in age from 59 to 83 years. Sixteen control cases were studied for an equal period of time (ten days). The cataract cases eliminated about 5 percent less of the acid than the controls. Several patients gained in weight, appetite, and general health, but definite gains in visual acuity were not noted. The author subscribes to the view that cataract patients suffer from avitaminosis C. The literature of the subject is reviewed to date.

Eugene M. Blake.

Butler, T. Harrison. **The anterior capsule of the lens.** Trans. Ophth. Soc. United Kingdom, 1939, v. 59, pt. 1, p. 3.

A detailed description of the embryology, histology, and clinical and pathological changes in the anterior capsule of the lens is given. The discussion includes anterior capsular, anterior polar, pyramidal, and posterior polar cataract, and also aftercataract. (45 illustrations.) Beulah Cushman.

Cashell, G. T. W., and Kon, S. K. **Studies in lactose and galactose cataracts in rats.** Trans. Ophth. Soc. United Kingdom, 1939, v. 59, pt. 1, p. 199.

In their experimental work the authors compared the toxicity of lactose as present in milk with that of the isolated substance when incorporated in synthetic diets. It was found that dried separated milk supplemented by minerals was no less toxic than an artificial diet of similar composition, but that the high protein content of milk exerted a protective action. Lactose caused a slowly growing cataract developing in the nucleus and the cortex. Lactose in liquid-milk form produced a dense opacity under the anterior pole of the lens capsule. Galactose cataract commenced with a dense film of fine bubbles at the equator of the lens. The feeding of fresh or reconstituted liquid separated milk was defi-

nitely more injurious to the lens than the feeding of milk in dry form. Discontinuance of galactose feeding produced regression of the cortical cataract, the nuclear opacity remaining the same. Beulah Cushman.

Hilgartner, H. L. **Management of juvenile cataract.** Texas State Jour. Med., 1939, v. 34, March, pp. 770-773.

The author tabulates and discusses the conditions found and the results obtained in fifty eyes with juvenile cataract. He speaks of simple discission as the favored procedure, although removal of dense capsules sometimes becomes necessary later. W. H. Crisp.

Klein, M. **Survey of different methods of wound closure after cataract operation.** Trans. Ophth. Soc. United Kingdom, 1939, v. 59, pt. 1, p. 293.

The author presents a statistical study of the effect of wound closure on healing after cataract operation. He compares the conjunctival-flap section made with the Graefe knife, the Kuhnt apron, and the corneoscleral suture according to Liégard. Results in 650 cataract cases as to duration of hospital treatment, visual acuity, postoperative astigmatism, and complications observed in the course of healing are given. It was concluded that visual acuity was worse after the Kuhnt apron operation but that the postoperative astigmatism was less. With the corneoscleral suture fewer days were spent in the hospital, but postoperative hemorrhages were more frequent than with the other methods.

Beulah Cushman.

Maisel, C. D. **A case of bilateral isolated coloboma of the lens.** Viestnik Opht., 1939, v. 15, pt. 2, p. 96.

In the left eye the coloboma was almost one fifth the normal size of the lens, and divided the pupil into aphakic and normal parts. The presence of opacities in the lens suggested an inflammatory process as the basis of the anomaly. Ray K. Daily.

Markin, L., and Keyes, P. **Species specificity of proteins of the optic lens.** Jour. Infect. Dis., 1939, v. 65, Sept.-Oct., pp. 156-159.

Lens proteins of widely different species present very similar antigenic properties, as determined by precipitin reactions. Using anaphylaxis as the immune reaction by which to compare lens proteins, results as published have been less uniform. The authors conducted a series of experiments using the anaphylactic reaction as a test of antigenicity. Pigeons sensitized to beef lens went into shock when injected with dog lens and vice versa. Shock from the injection of one lens protein desensitized the pigeons to the other. The sensitization of pigeons by injection of pigeon lens was unsuccessful, although guinea pigs demonstrated an anaphylactic response after autosensitization. The experimental results based on the anaphylactic reaction are in accord with those repeatedly observed with precipitin tests.

John C. Long.

Rubino, A. **Cataract and scleroderma (Rothmund-Werner syndrome).** Rassegna Ital. d'Ottal., 1939, v. 8, Sept.-Oct., p. 527.

The combination of cataract and scleroderma in early life was first noted in 1868, when five cases were reported in one family by Rothmund. Werner, in 1904, defined the skin changes as a true scleroderma. Only 35 cases have been recorded in the literature. The disease usually occurs between twenty and

thirty years of age, and 70 percent of the cases have been in women. It progresses slowly with general disturbances, such as loss of weight, appetite, and strength; sexual derangements; changes in the skin; and voice alterations. The skin changes are characterized by scleroderma, hypertrichosis, and melanosis. The cataract is always bilateral, develops fairly rapidly, and affects principally the posterior cortical layers. An endocrine disturbance is thought to be the basic etiologic factor. The author describes a typical case seen in a man of 38 years, whose sister and one brother were similarly affected. (4 figures.) Eugene M. Blake.

Stocker, F. **Retention of vision during development of senile cataracts.** Schweiz. med. Woch., 1939, v. 69, Oct. 28, p. 1037.

On the basis of observations of ten patients, Stocker urges that during the development of senile cataract improvement of vision may be brought about by removal of all sources of optical error in the media; a narrow gap in the lens opacity often giving opportunity for such improvement in vision.
Theodore M. Shapira.

10

RETINA AND VITREOUS

Bedell, A. J. **The fundus changes in two hundred and five diabetics.** Trans. Ophth. Soc. United Kingdom, 1939, v. 59, pt. 1, p. 219.

The fundus changes in 205 diabetic patients were analyzed and segregated in serial photographs, and their clinical and prognostic value emphasized. The number and size of hemorrhages and exudates were studied in the pictures and during the progress of the lesions.
Beulah Cushman.

Böck, Josef. **Clinical proof of adhesion of the vitreous body to the edge of the tear in detachment of the retina.** Graefe's Arch., 1939, v. 140, pt. 3, pp. 468-481.

The author reviews the literature concerning the origin of tears in retinal detachment. Histologic investigations and clinical studies (with Lindner's angled microscope, Koeppe's contact glass, and the slitlamp) show frequent separation of the vitreous from the detached retina, both before and after operation. The author discusses the papers dealing with attachment of the vitreous to the edge of the retinal tear. In the clinical study only cases with a far-posterior retinal tear can be used. Two such cases are described in detail. Each revealed the presence of posterior detachment of the vitreous, and its adhesion to one edge of the retinal tear. After reattachment of the retina with high-frequency current and penetrating needle-electrode, the vitreous was seen to be fused with the retina at the site of the scarred hole; the detachment of the vitreous posterior to this region was not altered by the operation. The second case showed preoperatively a detachment of the vitreous except at one point, the edge of the retinal tear. This suggested that the vitreous adhesion to the retina played a part in the formation of the retinal hole. The writer discusses the etiology of the retinal hole in detachment of the retina, emphasizing Lindner's theory of vitreous adhesion and Vogt's belief in the degenerative causation of retinal tears and holes. The ideas of previous workers concerning causes of adhesions between vitreous and retina, and especially the effects of myopic and senile degenerative changes and the results of inflammations involving the retina, are reviewed. Charles A. Perera.

Crecchio, A. de. **The retinal interchange after compression of the bulbus.** Ann. di Ottal., 1939, v. 67, Oct., p. 739.

The author studied the anaërobic glycolysis and the amount of respiratory exchange in the rabbit retina after compression of the globe for 30, 60, and 180 minutes. The tests were made 2, 5, 10, and 15 days after the compression was applied. The amount of oxygen consumed was diminished after compression lasting one to three hours. The author believes that the lessening of oxygen consumption was mechanical in character, and was due to retinouveal ischemia. (Bibliography.)

Park Lewis.

Ferguson, W. J. W. **Bipolar electrolysis in the treatment of detachment of the retina.** Trans. Ophth. Soc. United Kingdom, 1939, v. 59, pt. 1, p. 307.

The author describes the method of applying electrodes to the scleral surface after accurate localization of the retinal holes. The electrodes are applied for six seconds using six milliamperes of current, and enough applications are made to surround the retinal lesions. After electrolysis the subretinal fluid is released by ignipuncture or single incision with the Graefe knife and the conjunctiva is sutured. In 16 out of 25 cases the retina had remained replaced to the time of the report.

Beulah Cushman.

Fronimopoulos, J. **A contribution to the clinical study of detachment of the vitreous.** Graefe's Arch., 1939, v. 140, pt. 3, pp. 482-496.

The Gonin-Lindner theory of detachment of the vitreous as a frequent cause for retinal detachment is supported by the author's study of 26 patients with bilateral myopia and unilateral detachment of the retina. Detachment of the vitreous was found in 96 percent of the eyes with retinal separation and in 81 percent of the eyes with undetached retinas. The report of a case of retinal detachment in which the detached vitreous was adherent to the anterior edge of a retinal tear, and an instance of detachment of the retina developing in an eye with a previous vitreous separation further supports the Gonin-Lindner theory.

The second part of this paper deals with follow-up studies of the vitreous in patients with various types of ocular disease. Vitreous changes (development or increase of detachment of this structure) were not observed in eyes with tapetoretinal degeneration or healed chorioretinitis, but were found in eyes containing senile alterations of the fundus (12 percent) and in myopic eyes (29 percent of eyes with retinal detachment and 25 percent of detachment-free eyes). Changes in the vitreous body occurred in 75 percent of the eyes with retinal detachment following unsuccessful operation and in only 15 percent of such eyes after successful operative reattachment of the retina. The writer accounted for these findings by the presence of a passage through the retinal hole between the vitreous space and the subretinal fluid, the production of fluid by the ciliary body, and the absorption of fluid by the choroid. Charles A. Perera.

Magitot, A. **Pseudotumor (disciform) degeneration of the macula.** Ann. d'Ocul., 1939, v. 176, Oct., pp. 721-738.

A review of the literature and detailed histologic description of a case.

Redslob, E. **Macular lesions in young people.** Ann. d'Ocul., 1939, v. 176, Oct., pp. 738-744.

These macular lesions in apparently

healthy young people are not to be confused with the well-known hereditary macular degenerations or the cystic macular degeneration described by Vogt. They are the result of small macular hemorrhages of unknown etiology. The hemorrhages are not very destructive and cause relatively little visual disturbance. They do not produce extensive central scotomata and do not affect the color sense. The hemorrhages absorb completely, leaving only very small, almost imperceptible, chamois-colored plaques, with a few small scattered pigmented or yellowish points. John M. McLean.

Salus, Robert. **The vascular system of the fovea centralis.** Acta Ophth., 1939, v. 17, pt. 3, p. 279.

The author refutes the established conception of the avascularity of the fovea centralis, and of the choroidal nutrition of the outer retinal layers. This fallacious conception has its basis in anatomic studies of guinea-pig eyes. Salus holds that the structure of guinea-pig eyes and of human eyes is not identical. He describes the delicate and complicated vascular system of the fovea, as seen ophthalmoscopically in people under forty years of age. Because of the presence of the foveal vascular system and on the basis of clinical phenomena, he concludes that the entire retina is nourished by retinal blood vessels. The pathologic changes in the retinal vessels in arteriosclerosis involve first the foveal vascular system, and the earliest changes are to be found there. (Illustrations.) Ray K. Daily.

Waldman, Joseph. **Moving-picture record of the asteroid bodies in a case of asteroid hyalitis.** Amer. Jour. Ophth., 1940, v. 23, Feb., pp. 192-194.

11

OPTIC NERVE AND TOXIC AMBLYOPIAS

Evans, P. **Tobacco amblyopia.** Trans. Ophth. Soc. United Kingdom, 1939, v. 59, pt. 1, p. 241.

The author states that a definite diagnosis of tobacco amblyopia depends on the demonstration by screening of a centrocecal scotoma; that the primary etiologic factor appears to be a toxic agent derived possibly by a process of fermentation of tobaccos, present in higher amounts in the heavier and darker forms. The liver is involved in the detoxication process of the toxic agent. All factors of vascular sclerosis increase the probability of the onset of tobacco amblyopia; and the factor of vasospasm cannot be excluded. Alcohol in the form of beer has no effect on the incidence, severity, or rate of recovery. Beulah Cushman.

Hilgartner, H. L. **Benign cyst of the optic disc.** Amer. Jour. Ophth., 1940, v. 23, Feb., pp. 186-187.

Lauber, H. **Pallor of the disc.** Trans. Ophth. Soc. United Kingdom, 1939, v. 59, pt. 1, p. 231.

Pallor of disc may be due to thinning of the layer of nerve fibers and capillaries, to a change in the color of the blood, to disappearance of the blood in the capillaries, or to formation of new glial tissue on the disc. In lipemia the disc is very light in color and in ischemia the pallor may be due to glaucoma, quinine poisoning, or spasm of the central artery. The author presents his theory that the formation of tabetic nerve atrophy is associated with general hypotonia and disproportion of retinal pressure to intraocular tension. He also states that pallor of the disc

should not be confused with atrophy of the disc Beulah Cushman.

Lundberg, Åke. **A study on the development of oligodendroglia, astroglia, and the glial tissue of the optic nerve in fetal life.** Acta Ophth., 1939, v. 17, pt. 3, p. 259.

Report of microscopic study of 19 embryos in various stages of development, and of the optic nerve and chiasm of an infant at term. The sections demonstrated the development of the glial and mesodermal tissues of the optic nerve. The neuroglial cells are developed from ectoderm which forms the optic peduncle. At first undifferentiated cells, they appear as two separate types after penetration of centripetal fibers into the optic nerve; one type resembles the astrocyte and the other the oligodendrocyte. During the period of differentiation of the neuroglial cells, mesodermal elements enter the optic nerve from its sheath and advance along the capillaries in the nerve. As the mesodermal tissue divides the optic nerve into small bundles, the neuroglial cells assume their final form. (Photomicrographs.) Ray K. Daily.

Moore, J. E., and Woods, A. C. **Syphilitic primary optic atrophy. 2. General considerations and the results of treatment by standard methods.** Amer. Jour. Ophth., 1940, v. 23, Feb., pp. 145-157.

Robitashvili, K. F. **Involvement of the optic nerve in influenza.** Viestnik Opht., 1939, v. 15, pt. 2, p. 79.

A report of six cases of primary optic neuritis following afebrile and ambulatory cases of grippe. All recovered useful vision, so the prognosis in such cases may be regarded as fair.

Ray K. Daily.

Wolff, Eugene. **Some aspects of the blood supply of the optic nerve.** Trans. Ophth. Soc. United Kingdom, 1939, v. 59, pt. 1, p. 159.

The author describes the vascular supply of the optic nerve and emphasizes the fact that it resembles that of the brain. In the intracranial portion the pial network becomes thinner, and any interference with the blood supply would affect first and most markedly the central papillomacular bundle. The central retinal artery makes five bends as it enters the optic nerve and as it passes forward. The region of the lamina cribrosa is supplied by the circle of Zinn with branches from the collateral arteries. Beulah Cushman.

13

EYEBALL AND ORBIT

Delanoë, E. **Hydatid cyst of the orbit.** Ann. d'Ocul., 1939, v. 176, Nov., p. 828.

A report of a case of orbital tumor in a four-year-old boy from Morocco. The eye was enucleated and the orbit found to be filled with a large hydatid cyst.

John M. McLean.

Trovati, E. **An exploration of the ocular sympathetic-parasympathetic system in morbus Basedow's disease.** Ann. di Ottal., 1939, v. 67, Oct., p. 757.

Experiments were conducted on the ocular sympathetic and the parasympathetic systems, with a view to determining whether subjects of tachycardia exophthalmica were more easily affected by mimetic substances introduced in the lid fornix of the conjunctiva. To that end adrenalin and cocaine were employed. Trovati concludes that in monocular morbus Basedow's disease an equilibrium has been established in the regional neurovegetative system, and the same phenomena are

observed as in normal individuals. (Bibliography.) Park Lewis.

Varshavskaja, P. P. **Echinococcus cysts of the orbit.** Viestnik Opht., 1939, v. 15, pt. 2, p. 82.

A report of two cases. The unusual features of one case were the development of a fistula in the temporal region of the orbit, and a mastoiditis. The second case had symptoms of frontal-lobe involvement and otitis media, all of which cleared up after the removal of the orbital cyst. Ray K. Daily.

14

EYELIDS AND LACRIMAL APPARATUS

Busacca, Archimede. **Trachomatous nodules in pretarsal connective tissue and in the orbicularis.** Folia Clin. et Biol., 1939, v. 11, no. 6, p. 164.

In many trachoma cases, after the conjunctival process had been clinically cured, Busacca saw nodules in the pretarsal connective tissue and in the fibers of the orbicularis. Histologically he found nodules of lymphocytic infiltration, highly vascularized. Some of these nodules had a typical germinative center, and the vessels showed a cuff of infiltration. There was infiltration of the connective tissue separating the muscle fibers, with secondary atrophy of the muscular tissue. Some of the nodules gave signs of activity, and the author suggests that this may be the reason why some cases of apparently cured trachoma develop recurrences. (4 figures.) Eugene M. Blake.

Hadley, H. G. **Blepharochalasis.** Acta Ophth., 1939, v. 17, pt. 3, p. 310.

The literature is reviewed and a case reported of a 17-year-old boy who had typical recurrent attacks of edema of the lids for a period of 14 years, with bilateral skin changes and unilateral ptosis. Ray K. Daily.

Hambresin, L. **Radiotherapy of furuncles and hordeola.** Ann. d'Ocul., 1939, v. 176, Nov., pp. 803-812.

Hambresin strongly advises X-ray therapy for styes and furuncles of the face which are resistant to ordinary treatment. The danger of sinus thrombosis in cases of furuncle of the face is reviewed and X-ray therapy as the safest and the most satisfactory treatment is outlined. Early treatment with relatively weak and infrequent doses is recommended. Filtered X rays with low penetration and about 10 percent of the skin erythema dose are advised and the technical details are discussed briefly. There is a short review of the historical aspects with a brief discussion of the various theories as to the mode of action of X ray on local inflammatory processes. This form of therapy is also suggested for acute dacryocystitis, orbital periostitis, and orbital cellulitis. John M. McLean.

Kurlov, I. H., and Milovidova, A. H. **The use of the crochet hook for suturing the mucous membrane in dacryocystorhinostomy.** Viestnik Opht., 1939, v. 15, pt. 2, p. 38.

The flattened and perforated end of a crochet hook makes a convenient needle for deep suturing in the Toti operation. (Illustration.) Ray K. Daily.

Lipovskaja, A. I. **Gummatous involvement of the conjunctiva and lids.** Viestnik Opht., 1939, v. 15, pt. 2, p. 88. (See Section 5, Conjunctiva.)

NEWS ITEMS

Edited by Dr. H. Rommel Hildreth
640 S. Kingshighway, Saint Louis

News items should reach the Editor by the twelfth of the month.

Deaths

Dr. Webb William Weeks, New York, died January 10, 1940, aged 53 years.

Dr. Harry Milton Weed, Buffalo, died December 5, 1939, aged 65 years.

Dr. Daniel George Monaghan, Denver, Colorado, died January 17, 1940, aged 65 years.

Dr. T. H. Shastid of Duluth sends notice that his wife, aged 70 years, died suddenly of heart disease on March 1st.

Miscellaneous

The Cook County Graduate School of Medicine (in affiliation with the Cook County Hospital) announces continuous courses in ophthalmology and otolaryngology

Course No. 1 in ophthalmology is an informal course for observation of the routine of examination, diagnosis, and therapy as carried on daily in the out-clinic, ward, and operating room. The course is available, starting the first of every week, except when Course No. 2 is in progress. Fees: Two weeks, $100.00; One month, $175.00.

Course No. 2 is a two weeks' intensive clinical and didactic course covering, in condensed form, the present status of the most important phases of ophthalmology, with special emphasis on developments during the past decade.

The course will start on April 22d and September 23d if six doctors are registered two weeks in advance of the starting date. The enrollment will be limited to six doctors and registrations will be accepted in the order received. Fee $200.00.

The Harvard Medical School announces a course in Visual Optics and Physiology from July 1 to July 27, 1940.

This course, conducted by Drs. Ludvigh, Cogan, and Easton with the assistance of other members of the staff, is an all-day course given six days a week during the month of July. Lectures, laboratory work, and discussion occupy seven hours a day and outside reading is assigned covering articles on special subjects in the modern American and English literature.

Although the course is not designed primarily for beginners and has been taken by instructors in visual optics and physiology, past experience shows that due to a rapid first week review an ophthalmologist with diligent work may take the course to relatively greater advantage.

The first two weeks of the course cover approximately the range of material given in Duke-Elder's "Textbook of ophthalmology," volume 1, pages 483 to 1,086. Subsequently a treatment of more advanced topics not thoroughly discussed in the average English or American textbook is undertaken.

The executive secretary of the Missouri State Medical Association has forwarded a circular issued by Mr. D. W. McKinnon, Sheriff of Grundy County, Missouri, concerning a forger who plays upon oculists and optometrists. The man, who is about 50 years old, 5 feet 9 inches tall, weighs 160 pounds, has blue eyes, and light reddish-brown hair. He presents himself for examination, requests bifocal glasses, and upon payment offers a check for 30 or 36 dollars made out to himself for produce sold by him.

To meet a nationwide need for medical personnel trained in aviation ophthalmology and aviation medicine, the George Washington University School of Medicine, through its Department of Ophthalmology, will present an intensive course in this field, April 1 to 6, 1940.

The course is part of the post-graduate course in ophthalmology given by the School of Medicine, which includes also a special practical course in ocular surgery, pathology, and orthoptics, limited to 25 participants, to be given March 19th to 23d, and an intensive post-graduate course in ophthalmology, March 25th to 30th.

Societies

The Sixth Australian Medical Congress to be held in Perth, Western Australia, in September, 1940, under the presidency of Dr. D. D. Paton, Honorary Ophthalmic Surgeon, Children's Hospital, has been postponed on account of the international situation.

The Eye Section of the Philadelphia County Medical Society presented the following program on March 7, 1940: Post-graduate conference in ophthalmology by Drs. Krewson, Spiegel, Olsho, and Spaeth. Case report by Dr. Carl Breisacher. Case of exophoria with bilateral over-acting inferior obliques by Dr. M. Luther Kauffman. Recent advancements in retinal detachment surgery by Dr. James S. Shipman.

The Oxford Ophthalmological Congress will convene at Oxford on July 4 to 6, 1940, with

Prof. le Gros Clark delivering the Doyne Memorial Lecture.

The executive committee of the recently organized Detroit Ophthalmological Society announces the following officers for 1940: honorary presidents, Dr. Walter R. Parker and Dr. Don M. Campbell; president, Dr. Parker Heath; president-elect, Dr. Ralph Pino; secretary, Dr. Leland F. Carter; treasurer, Dr. Cecil Lepard; recorder, Dr. Edmond Cooper. Active members, 39. Membership requirements, American Board of Ophthalmology certificate. Meetings to be held the third Thursday of each month from November to April, inclusive. Club rooms of the Wayne County Medical Society.

Personals

Dr. Orwin H. Ellis announces the opening of his office, 514 Wilshire Medical Building, Los Angeles.

Mr. H. L. Eason was recently elected president of the General Medical Council, London.

Mr. H. M. Traquair was recently elected president of the Royal College of Surgeons of Edinburg for the ensuing year.

Dr. I. Barraquer has recently announced the removal of his clinic to a new building at 314 Calle Muntaner, Barcelona, Spain.

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